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ON THE CAUSES OF CARDIAC INSUFFICIENCY.[1]

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Bamberger[2] was the first to recognize that true cardiac insufficiency is a muscle insufficiency. It is an inability of the heart muscle to do its work. Cardiac insufficiency is, of course, not synonymous with valvular insufficiency. The healthy cardiac muscle by doing extra work, usually compensates for the regurgitation caused by a leaky valve.

The work done by the heart is determined by the quantity of blood which enters the heart during diastole and the amount of resistance to be overcome in forcing the blood into the arteries. Pathological conditions can only affect the work of the heart by altering the resistance of the diastolic blood-quantum.

In cardiac insufficiency there is a disproportion between the heart's capacity for work and the amount to be done. It does not matter whether the working power of the heart be diminished so that it is unable to meet normal demands or whether the demands made upon a healthy heart are so increased that its functional capacity is inadequate. In either case cardiac insufficiency is the result.

The cardiac incompetency is due either to the inability of the heart to dilate sufficiently to receive all the blood that flows from the veins, or the inability to propel the blood through the arteries with proper velocity. Hence the fault lies either in a diminution of the heart's power of contraction or its power of dilatation.

If the weakness affects chiefly the left ventricle,[3] less blood will be thrown at each systole into the aorta. The arteries will be but scantily filled and hence there will be a diminution in the size of the pulse and a fall of arterial blood pressure. The feeble ventricle will contain at the end of systole the blood it was unable to eject into the aorta and this will prove a hindrance to the entrance of blood from the left auricle during diastole. The left auricle will become distended, and congestion of the pulmonary vessels will result.

If the right heart is able to overcome the increased resistance, the blood pressure in the lung will be abnormally high. The right ventricle will eject no more blood than the left ventricle, because it receives no more. The circulation of the blood is slowed, the distribution altered and the lungs overfilled. If the right heart weakens then the blood will collect in the right auricle and venous trunks, and if the incompentency is marked chronic passive congestion of all the ab-

[1] A lecture delivered in the course for graduates at the Johns Hopkins Medical School.

[2] Bamberger: Lehrbuch der Krankheiten des Herzens. Vienna, 1857, p. 313.

[3] Compare Cohnheim, Lectures on General Pathology. Trans. by McKee, London, 1889, 1. p. 78, v. Basch, Allgemeine Physiologie und Pathologie des Kreislaufs, Vienna, 1892, p. 103. Krehl, Pathologische Physiologie, Leipsic, 1898, p. 64. Moritz, Deutsches Archiv für klin. Med., 1899, LXVI, p. 349. Lazarus-Barlow, Manual of General Pathology, 2nd edit., London, 1904, p. 77.

dominal organs will ensue. With weakness of the right ventricle the arteries of the systemic circulation are, as a rule, less well filled than normally and the blood pressure is low, even if the left heart is relatively strong. This is easily explained, the left heart receives less blood during diastole and hence the amount injected during systole must be diminished. Usually cardiac insufficiency affects both ventricles.

If the heart does not contract completely there will remain at the end of systole an increased amount of blood in the ventricle. This, added to the blood entering the heart during the next diastole, distends the cavity and the condition is spoken of as dilatation. There are all degrees of cardiac insufficiency from the slight disturbances of function, only apparent after severe muscular efforts, to completely broken compensation.

In many cases of acute and chronic disease the issue between life and death rests with the heart. Speaking of pneumonia in the aged, Huchard says *"La maladie est au poumon, le danger est au coeur."*

The condition of the heart muscle determines the clinical course of chronic valvular disease. The cause of the great variations in the clinical pictures depends less on the valvular defect than on the condition of the cardiac muscle. The nature and severity of the valvular lesion alone will not explain the circulatory disturbances. A patient with marked mitral insufficiency as determined by physical examination may be able to climb a high flight of stairs or do considerable work without becoming short of breath. Another patient with a less degree of the same valvular lesion may not be able to take a dozen steps and be quite incapable of doing any bodily work.[4]

The contraction of the cardiac muscle cannot be compared to that of the biceps. As Martius[5] says the heart is not a hollow muscle but a hollow muscular organ. It works continuously and it does not become exhausted. With each systole it does .2 to .8 kgm. of work, and according to Zuntz[6] drives forward sixty cubic cc. of blood and maintains an arterial blood pressure of about 160 mm. of mercury. The work done by the heart in a day amounts to about 75,000 kilogrammemeters, which Foster[7] estimates is just about the amount of work done in the ascent of Snowden by a tolerably heavy man.

The work of the heart at each beat may be increased in two ways. Either it has to pump more blood than usual or the resistance to the cardiac contraction is augmented. If an increased amount of blood enters the heart at each diastole more blood than normal must be forced out at each systole and, unless the frequency of the beats is diminished or the time of contraction increased, the heart must do more work. Experiments show that when the heart is unusually distended during diastole the frequency of the systolic contractions is not changed and hence the muscle fibres must contract more vigorously.[8] The reserve-power of the heart is enormous and so it meets the extra demands with ease. Lewy[9] has shown that the heart exerted to its maximum is capable of doing thirteen times as much work as it does when the body is at rest. By stimulating the vagus the diastole is prolonged and the heart becomes greatly distended with blood. Stolnikow[10] found that the next systole following stimulation of the vagus was able to propel more than six times the usual amount of blood. Also by pressure on the abdomen and by other means, the heart can be greatly distended with the same result. The pulse rate is not disturbed, the contraction of the heart is simply more powerful. This adjustment of the heart to varying quantities of the blood takes place so quickly that v. Frey maintains it cannot be due to reflex action. We know little in regard to the influence of the vagus, the accelerans, or the cardiac ganglion cells in the production of disturbances of cardiac function, and the significance of anatomical alterations in these structures is not understood. The researches of His[11] and Romberg[12] have shown that the ganglion cells are sensory, not motor in nature. It was demonstrated many years ago that if the nerves which pass to the heart were severed the power of accommodation was still preserved. This regulatory mechanism probably resides in the muscle fibres. There is no evidence to show that the cardiac muscle fibres do not work automatically,[13] and it may be that the greater stretching of the muscle fibres by the distention of the wall enables them to contract more vigorously.[14] Martin[15] showed twenty years ago that the mammalian heart removed from the body would beat for hours in a normal manner.

As the working power of the heart is dependent on the contractility of the muscle fibres and the elasticity of the heart wall, any cause which diminishes the contractility and the elasticity will tend to produce cardiac insufficiency. We shall now consider in some detail the anatomical alteration of the heart muscle which has generally been thought to be the most common cause of cardiac weakness, and that is fatty metamorphosis. The term fatty heart embraces two entirely distinct anatomical conditions. It refers both to an increase of adipose tissue between the heart fibres, and to a deposit of fat droplets within the muscle cells. The former condition is

[4] Vide Krehl: Deutsches Archiv f. klin. Med., 1890, XLVI, p. 454.

[5] Martius: Volkmann's Sammlung klinische Vorträge, 1894, No. 113, p. 192.

[6] Zuntz: Deutsche med. Woch., 1892, p. 109.

[7] Foster: A Text-book of Physiology, sixth edition. London, 1893, Part 1, p. 268.

[8] v. Frey: Deutsches Arch. f. klin. Med., 1890, XLVI, p. 398. Krehl: Pathologische Physiologie, Leipsic, 1898, p. 5.

[9] Lewy: Zeit. für klin. Med., 1896, XXXI, p. 321.

[10] Stolnikow: Du Bois' Archiv für Physiologie, 1886, p. 1.

[11] His: Verhandlungen des IX Congress für innere Medicin. Wiesbaden, 1890, p. 367.

[12] Romberg: Ibid, p. 356.

[13] Engelmann: Pflüger's Archiv, 1897, CLXV, p. 535. Engelmann: Die Deutsche Klinik, Berlin, 1903, IV, p. 215. Gaskell: Schäfer's Text-book of Physiology, London, 1900, II, p. 169.

[14] This is the view of v. Frey and Krehl. Roy and Adami showed (Phil. Trans. Roy. Soc., London, 1892, CLXXXVIII, p. 213) that distention increases the resistance to contraction.

[15] Martin: Studies from the Biological Laboratory of the Johns Hopkins University, 1883, II, p. 119.

usually called fatty overgrowth or fatty infiltration, and the latter fatty degeneration.

The fatty metamorphosis is regarded by many authorities as a most serious alteration in the structure of the heart. Broadbent[16] states that "no form of heart disease is regarded with so much apprehension as fatty degeneration. And when the diagnosis has once been made prognosis for the most part can contemplate only one result; the fatal termination is merely a question of time and circumstance." Gibson[17] who devotes much space to its consideration asserts that fatty degeneration of the heart is the most common cause of sudden death. Many American writers express similar opinions.[18]

It appears that Harvey in the seventeenth century published the first observation on fatty heart. Upon dissecting the body of a very corpulent man he found the heart completely covered with fat, "*Cor adipe plane tectum.*"

Both varieties of fatty heart were recognized by Corvisart[19] and differentiated and described by Laennec[20] but we owe the first careful anatomical study of this condition to the English pathologists, especially Quain,[21] and to Rokitansky.[22]

One of the first recorded cases of fatty degeneration was that studied by John Cheyne[23] in 1818. At that time fatty degeneration of the heart was regarded as very rare. Corvisart,[24] stated that he had never seen a case. Cheyne's paper appeared in the second volume of the Dublin Hospital Reports. The case is of historical interest not only as being probably the first carefully recorded instance of fatty metamorphosis of the heart but also the first case in which the peculiar modification of respiration, now known as the Cheyne-Stokes was described. This type of breathing was thought by Stokes to be almost pathognomonic of fatty heart. The patient was sixty years of age. His pulse was irregular and unequal. There was œdema of the ankles in the evening. He had one attack of unconsciousness not followed by paralysis, but shortly before his death he became hemiplegic. At autopsy the brain showed merely congestion. The note on the heart was as follows: "The lower part of the right ventricle was converted into a soft fatty substance. The upper part was remarkably thin, and it gradually degenerated into this soft, fatty condition. The cavity of the left ventricle was greatly enlarged, and its whole substance with the exception of the internal reticulated structure and carneæ columnæ was converted into fat. The valves were sound, and the aorta studded with steatomatous and earthy concretions." No mention was made of the coronary arteries.

Later Robert Adams[25] published a case which Stokes said furnished the key to the diagnosis of this condition. This report like the one I have just quoted is of unusual interest, because it contains the first account of the Stokes-Adams syndrome. That, you remember, is the association of a persistently slow pulse with attacks of unconsciousness. The patient's age was sixty eight. The heart was very fatty, the condition of the coronary arteries is not described but the statement is made that "the coats of the carotid and middle arteries of the dura mater were quite white and opaque from bony deposits."

Rokitansky studied fatty metamorphosis of the heart microscopically and stated that the fat occurred as minute globules imbedded within the muscle among the primitive fibres, the transverse striæ of which were lost.

The conception that the cell proteids degenerated into fat dates from Virchow's article published in the first volume of his Archiv.[26] Two forms of fatty alterations of the parenchymatous tissues were recognized. Fatty infiltration in which the fat was deposited in the cells, and fatty degeneration of the cells. It was known that fat brought by the blood was normally taken up and deposited in the cells of the liver and in the connective tissue cells, especially those of the subcutaneous and subserous tissues. This was described as physiological fatty infiltration. If fat were deposited in great excess in these structures, for example in the liver, or if it were deposited in connective tissues, which normally contained little or no fat, it was spoken of as pathological fatty infiltration. When fat appeared in cells in which it was not normally demonstrable by microscopic methods, as those of the heart and kidney it was regarded as a fatty degeneration. It was taught that the diagnosis could be made histologically. In fatty degeneration it was held that the fat appeared in minute droplets which did not tend to become confluent, while in fatty infiltration the drops coalesced and pushed the nucleus to one side. This histological distinction was admitted not to be applicable to all cases. The fatty change in the kidney was regarded as a degeneration although the fat appears not only as minute granules but as large drops. Hansemann,[27] however, within the last few years came to the conclusion that fatty infiltration of the kidney could occur. The fatty liver resulting from phosphorous poisoning was regarded as a typical example of fatty degeneration and yet the fat appeared in the form of large as well as small drops.

The fatty degeneration was regarded as a very serious alteration in the cell. According to A. Fraenkel,[28] there was first

[16] Broadbent: Heart Disease. New York, 1897, p. 282.

[17] Gibson: Diseases of the Heart and Aorta. Edinburgh, 1898, p. 649.

[18] Forchheimer: Trans. Assoc. Am. Phys., 1888, III, p. 89. Robinson: Am. Jour. Med. Sci., 1901, CXXI, p. 151. Anders: Ibid., 1901, CXXI, p. 429. Babcock: Diseases of the Heart and Arterial System, New York, 1903, p. 518. Warthin: Wood's Reference Handbook of the Medical Sciences, New York, 1902, IV, p. 589. Satterthwaite. Medical News, 1901, LXXVIII, p. 165.

[19] Corvisart: An Essay on the Organic Diseases and Lesions of the Heart and Great Vessels. Trans by Gates. Boston, 1812, p. 153.

[20] Laennec: A Treatise on Diseases of the Chest. Trans. by Forbes, 1838, p. 682.

[21] Quain: Med. Chir. Trans. London, 1850, XXXIII, p. 121.

[22] Rokitansky: A Manual of Pathological Anatomy. London, 1852, IV, p. 204.

[23] Cheyne: Dublin Hospital Reports, 1818, II, p. 221.

[24] Corvisart: Loc. cit., p. 154.

[25] Adams: Dublin Hospital Reports, 1827, IV, p. 353.

[26] Virchow: Virchow's Archiv, 1847, I, p. 94.

[27] Hansemann: Virchow's Archiv, 1897, CXLVIII, p. 355.

[28] Fraenkel: Virchow's Archiv, 1876, LXVII, p. 273.

a kind of necrobiosis of the cells, and that the cell proteids were split into a nitrogenous and non-nitrogenous moiety, and from the non-nitrogenous moiety fat was formed. It was but natural to assume that the function of a heart, the seat of such a destructive process, must be seriously impaired.

It appears that the idea of Virchow that the proteids of diseased cells become converted into fat was accepted without much question, although no proof was adduced. Later it was supported by the experiments of Voit who claimed that animals fed on a pure meat diet took on fat. He concluded that nearly all the body fats were formed from proteids. Pflueger later showed that there where errors in Voit's analyses and that the conclusions were unwarranted.

There have been two periods, as Fischler[20] points out, in which especial interest has been manifest in regard to the significance of fat in its relation to pathological processes.

The first of these periods was in the middle of the last century, and dated from the appearance of Virchow's paper. Following the publication of Virchow's views a great deal was written on fatty degeneration, but very little original work was done.

The second epoch began in 1897, with the report made by Georg Rosenfeld,[20] of Breslau, at the German Congress of Internal Medicine. Pflueger had previously showed that the fat formed in the ripening of cheese and in adipocere was due solely to the action of bacteria, and hence was not analogous to the formation of fats in the living animal body. It was known that if a dog were starved for a number of days and then given phloridzin that the liver would be found extremely fatty. Now, if this fat, said Rosenfeld, which may amount to 75 per cent. of the weight of the dried liver, is formed from the cell proteids, as the pathologists teach, a great part of the cell proteids must be destroyed in the production of this amount of fat. But he found by examination that very little proteid had been lost, hence the fat must have been introduced from without. This explanation is supported by the fact that the blood of animals poisoned by phloridzin is rich in fat. The cells of the liver show no signs of degeneration and if the dog is starved again after the administration of phloridzin the fat disappears and the organ takes on its normal appearance. He demonstrated that the fat was brought from the subcutaneous tissues and paraperitoneal spaces by the following experiment. A dog was deprived of its fat by long starvation and then fed with a large amount of mutton suet. In the ordinary fat depots including the liver, mutton suet was found deposited. A few days without food, however, caused the fat to disappear from the liver, as was shown by control animals. Now animals thus treated had fat depots loaded with mutton suet, but livers free from fat. If Rosenfeld's view were correct the administration of phloridzin would cause mutton suet to wander from the fat depots to the liver and a mutton suet liver would result, and thus it proved; the animals so treated had 50 per cent. of mutton suet in the liver.

As we have said, the typical example of fatty degeneration of the liver cited by pathologists is that seen in phosphorous poisoning. If the fat is formed from the cell proteid, Rosenfeld reasons it will make no difference whether the fat storehouses are rich in fat or not, but if on the other hand the fat is brought to the liver from the fat store-houses, there can be no fat liver if the fat depots contain only a scanty supply of fat. And this is what he found. Very lean dogs poisoned with phosphorus did not exhibit fatty livers while in well nourished dogs the fat liver appeared. Dogs whose fat deposits were filled with mutton suet were given phosphorus and starved. When killed the livers were markedly fatty but the fat was mutton suet. Rosenfeld also showed that the fat in milk was not formed from the proteids in the mammary gland but was derived from fat which was brought from the fat store-houses to the breast.

Others[21] have confirmed and amplified the work of Rosenfeld. Many physiologists look upon the formation of fats from proteids not only as unproved but as improbable. Some believe that it may occur, although they do not regard it as an important mode of origin. It was known that the mammary gland and liver were concerned in the metabolism of fat, but it was different with organs such as the heart which do not store up fat normally. It did not follow from Rosenfeld's experiments that fat in the heart was not derived from a degeneration of its protoplasm. Rosenfeld in 1901,[22] however, and Leick and Winckler in 1902,[23] found that the fatty degenerated hearts of dogs which had been fed on mutton suet and then poisoned with phosphorus contained a fat identical with mutton suet. This proves that the so-called fatty degeneration of the heart is really another example of fatty infiltration.

The work of these investigators is important in regard to the clinical significance of fatty heart. So long as it was thought that the fat in the muscle cell was formed from the cell proteids it seemed as if the function of the cell must be seriously disturbed, although clinical observations, as we shall see, did not support this view. But if the fat is simply deposited in the cell it is different. We know that the protoplasm of the liver cells can appear microscopically to be almost entirely replaced by fat, and yet the functions of the organ be slightly if at all disturbed. The fatty liver of chronic alcoholism is an example of this. It has been shown that the fatty lesion is not due to a deficient supply of oxygen to the cells as was formerly held. It is possible that the fat is deposited in cells injured by the action of toxins. The fat

[20] Fischler: Virchow's Archiv, 1902, CLXX, p. 100.

[20] Rosenfeld: Verhand. des XVI Congr. für inn. Med., Wiesbaden, 1897, p. 427.

[21] Kraus und Sommer: Hofmeister's Beiträge, 1902, II, p. 86. Leick und Winckler: Arch. für exp. Path. u. Pharm., 1902, XLVIII, p. 163. Rosenfeld: Ergebnisse der Physiologie, Wiesbaden, 1902, I, p. 651. Ibid., 1903, II, p. 50. Herxheimer: Erbegnisse der allgemeine Pathologie, Wiesbaden, 1904, p. 625. Kraus Cent. f. allg. Path., 1904, XV, p. 41.

[22] Rosenfeld: Cent. f. inn. Med., 1901, p. 145.

[23] Leick und Winckler: Loc. cit.

may wander into the degenerated protoplasm of the heart muscle to fill up a vacant space just as connective tissue will grow into an area of necrosis and replace the destroyed cells. Leick and Winckler also suggest another possibility. The fat does not become visible in the healthy heart muscle because as fast as it is taken up by the protoplasm it is used to supply energy to the cell. The cardiac muscle takes up no more than is needed for immediate use, but a damaged cell may not be able to use the fat which it ingests in normal amount and hence the fat accumulates in the cell and becomes visible. Or possibly the fat enters the injured cell more readily and in larger amount. The fat in a cell occurs as free fat and as fat combined with protein. The combined fat is not visible. Taylor,[54] found that in fatty degeneration the combined fats were diminished and it may be that the fat visible in the muscle fibres of the heart is formed in part from the combined fat by autolysis. On this point, however, we have no exact information. Tissues which are microscopically fat-free and appear normal have been found on chemical analysis to contain as much as 20 per cent fat. On the other hand a pathological kidney with a slight content of fat can appear markedly fatty on microscopical examination.[55] There is evidence to show that fat is not deposited in dead cells.[56] Fat in a cell, Rosenfeld says, is a sign of life, not of death. Hester[57] and Hagemeister,[58] believe that disturbances of circulation bring an increased amount of fluids containing split fats, and these within the cell by a synthetic process are converted into neutral fats.

We can conclude from the result of this recent work that although the formation of fats from proteids in the animal body cannot be denied it has not been proved.

We will now inquire into the frequency, location, and amount of visible fat in the heart.

The frequency with which the heart muscle cells are the seat of fatty metamorphosis is not generally recognized. Dr. Osler[59] states that it is a common condition, but the majority of clinical writers imply that it is rather rare except in chronic heart disease and in severe anæmia and intoxications. It was a routine procedure for several years in Dr. Councilman's laboratory to examine a fresh specimen of the heart muscle for fat at every autopsy. My interest in the subject was aroused, not only by the frequent occurrence of marked fatty degeneration but also because no relation seemed to exist between fatty heart and cardiac weakness. We found that extreme fatty metamorphosis could be present without producing symptoms and in some cases in which cardiac weakness had been prominent the myocardium contained little or no fat. Osler[60] says in his text-book that extreme fatty changes, as in pernicious anæmia may be consistent with a full regular pulse, and a regularly acting heart. In some of these cases the fat does not appear to interfere seriously with the function of the organ.

Until the microscope came into use fatty degeneration of the heart was regarded as rather uncommon. You recall the traditional description of the heart, the seat of fatty degeneration. It is stated to be soft and flabby, with dilated cavities and of a pale yellow color. The endocardium, especially over the papillary muscles often presents a mottled appearance compared to a faded leaf, a tiger lily or a thrush's breast. The heart of pernicious anæmia usually answers to this description exactly. Our routine examinations showed, however, that a heart might be the seat of extensive fatty degeneration, and yet be firm, contracted, and of normal color, and without mottling of the subendocardial layer. The consistence depends largely on whether the heart stopped in systole or diastole and on the presence or absence of rigor mortis, while the yellowish hue is due in good part to the associated anæmia. And if the fat is uniformly distributed throughout the muscle the mottling which is caused by the localized deposition of fat will not appear. A heart which contains very little blood but no fat has a yellowish hue and may easily be mistaken for a fatty heart. Wagner called attention to this source of error.

Nearly all the well-known treatises on heart disease contain the statement that fatty degeneration is frequent in the hypertrophied hearts of chronic valvular disease and in myocarditis. And they lay emphasis upon its importance in the causation of disturbances of compensation. Wagner said that fatty degeneration is found in one-third of the cases of valvular disease. More recent writers (v. Recklinghausen, Zenker, and Krehl[61]), assert that fatty degeneration is rare in chronic heart disease and Bollinger came to the same conclusion in regard to the cases of cardiac weakness occurring among the Munich beer drinkers.

Very few observers have determined carefully the amount of fat present. The diagnosis has been made as we have said, either on the naked eye appearances or by examining microscopically one portion of the heart wall. The amount of fat present can be estimated either by a quantitative analysis or by a microscopic examination of all parts of the heart. Krehl adopted the chemical method; he removed the adipose tissue from the surface and substance of the heart and determined the quantity of fat in the entire organ. The dried substance of the normal heart contains about 8 per cent of fat. Only in phosphorus poisoning was marked increase of fat constantly found. In this condition about 25 per cent of the organ was converted into fat. In pernicious anæmia there was an average of 13 per cent found; a larger percentage than was present in any other disease except phosphorus poisoning. But there was no relation to be traced between the cardiac disturbance and the amount of fat present. The heart containing the largest quantity of fat had exhibited no signs of weakness while in another case in which there was dilatation of both ventricles, a systolic mitral murmur, and accentuated

[54] Taylor: Journal of Medical Research, 1903, IX, p. 59.
[55] Fischler: Virchow's Archiv, 1902, CLXX, p. 100.
[56] Kraus: Cent. für allg. Path., 1904, XV, p. 41.
[57] Hester: Virchow's Archiv, 1901, CLXIV, p. 293.
[58] Hagemeister: Virchow's Archiv, 1903, CLXXII, p. 72.
[59] Osler: Principles and Practice of Medicine. Fifth edition, New York, 1902, p. 749.
[60] Osler: Loc. cit., p. 750.
[61] Krehl: Deutsches Arch. f. klin. Med., 1893, LI, p. 416.

second pulmonic sound, the percentage of fat in the heart was practically normal. In cases of nephritis, malignant tumors, and tuberculosis there was some increase of fat (average 10 per cent), but the amount present in myocarditis and chronic valvular disease was within the limits of normal.

The estimation of the facts by the microscope, if carried out in a thorough manner, has certain advantages over the chemical method. The latter is open to the objection that all the fat present in the intermuscular tissue cannot be removed. But what is more important, the location of the fat, and the degree of destruction of contractile substance in the muscle is not ascertained.

Extreme deposition of fat in one part of the heart might indicate greater injury to its working power than a larger amount uniformly distributed. The stimulation for the systolic contraction arises in the muscle fibres of the auricles near the great veins. It is quite possible that a fatty change at this point, or in His' bridges, which transmit the contraction from auricle to ventricle, might impair the action of the heart while an equal fatty alteration elsewhere would not. Unfortunately no exact data are available on these points. So far as I know systematic investigations of the distribution of fat and the associated alterations in the contractile substance of the heart have not been made. I have recently adopted the method of examining teased specimens from 25 different portions of the myocardium selected as follows: Three examinations of each auricle; (1) near the orifices of the great veins; (2) near the auriculo-ventricular valve; (3) in the middle of the auricular wall. Seven examinations of the left ventricle; (1) near aortic ring; (2) near mitral ring; (3 and 4) Centres of anterior and posterior papillary muscles; (5) apex; (6) posterior wall, midway between base and apex, beneath pericardium, (7) posterior wall, midway between base and apex, center of muscle (8) posterior wall, midway between base and apex, beneath endocardium. Seven examinations of the right ventricle; (1) near pulmonary valve; (2) near tricuspid ring; (3 and 4) centres of two papillary muscles; (5) near apex; (6) anterior wall midway between base and apex, beneath pericardium; (7) posterior wall midway between base and apex, central muscle; (8) posterior wall midway between base and apex beneath endocardium. Three examinations of septum (1) beneath endocardium of right ventricle; (2) beneath endocardium of left ventricle; (3) in center of muscle.

The fresh tissue is teased out and treated with glacial acetic acid. Fat cannot be distinguished from the myelin droplets of Kaiserling,[1] by this method, but inasmuch as myelin droplets have never been found in the heart there is no valid objection to the use of acetic acid in this study. The use of Kolossow's method,[2] would prevent error, as myelin is only tinted a light gray by osmic acid.

In certain cases it might be desirable to examine ten or twenty-five additional portions of the muscle. A few bits of tissue showing the most marked fatty metamorphosis are treated by Kolossow's method.

The heart is then hardened in 10 per cent of formalin and a systematic examination of the structural alterations in the coronary arteries, muscle fibres, or interstitial connective tissue can be made by Krehl's method. The examination for fat can be continued by means of frozen sections stained with sudan III or scharlach R. The advantages of this method may be illustrated by a recent case in which there was profound anæmia resulting from purpura hæmorrhagica. The heart was large, weighing 440 gms., and there was marked dilatation of both ventricles and auricles. The typical picture of advanced fatty degeneration was presented. The heart was flabby, soft and yellow. The endocardium of the left ventricle mottled. The valves and coronary arteries appeared healthy. There was no arterio-sclerosis elsewhere and the kidneys were normal. A teased specimen of a papillary muscle attached to the mitral valve showed the muscle fibres were studded with fat. It certainly seemed in this case more than in any other of my series that weakness and dilatation were due to extensive fatty metamorphosis.

But the examination for fat made according to the method outlined above showed that only in the papillary muscles was the fatty change of a high degree. Very little fat was found except beneath the endocardium of the left ventricle. The middle and outer portions of the left ventricular wall the right ventricle and both auricles, were practically free from fat. Surely in this case cardiac dilatation was not due to fatty metamorphosis.

At the Boston City Hospital during the years 1898 and 1899, 477 autopsies were performed. In 120 of these the statement is made in the autopsy protocols that fat was found in the myocardium on examination of a teased specimen or frozen section. According to this, fatty metamorphosis occurred in 25 per cent of all the cases that came to autopsy during these two years. My friend Dr. H. A. Christian found fatty metamorphosis of the heart in 22 out of 27 consecutive cases examined. In nine of these the fatty change was marked. Only one portion of the myocardium, usually the wall of the left ventricle, or one of its papillary muscles, was examined. It is generally agreed that the papillary muscles of the left ventricle are the most common site of the fatty lesion.[3] We can draw no conclusion from these statistics in regard to the existence of the fatty change in other parts of the heart.

The presence of fat in moderate amounts may not be associated with apparent injury to the heart muscle. Virchow stated than even in well marked degeneration the primitive fibres were preserved. The contractile substance is known to be very resistant to autolytic changes.[4] And Ribbert[5] points

[1] Kaiserling und Orgler: Virchow's Archiv, 1902, CLXVII, p. 296.

[2] Kolossow: Zeit. f. wissen. Mikros., 1892, IX, p. 38.

[3] Cowan: Journal of Path. and Bact., 1902, VIII, p. 183.

[4] Dietrich: Cent. f. Path., 1904, XV, p. 84.

[5] Ribbert: Lehrb. d. allg. Path., Leipsic, 1901, p. 216.

out that mitotic figures can sometimes be seen in fatty degenerated cells.

Experimental researches fail to show that the presence of fat in the muscles interferes with the work of the heart. Dr. Welch,[47] found that in rabbits with marked fatty degeneration of the heart, the blood pressure and the reaction to vagus stimulation was normal. He observed rhythmical contraction in a group of muscle fibres filled with fatty globules. The degeneration was so advanced that only an indistinct trace of striation could be detected. Bálint[48] produced aortic insufficiency experimentally in dogs and then poisoned them with phosphorus, but no signs of cardiac weakness developed although the hearts were extremely fatty. Hasenfeld and Fenyvessy,[49] in a recent experimental study have shown that even marked fatty degeneration of the heart does not notably reduce its reserve power.

We have examined the evidence furnished by anatomical, chemical, and experimental investigations without finding any support for the theory that fatty metamorphosis of the heart is the cause of cardiac insufficiency.

The cases which Stokes[50] studied with such care and so graphically described certainly suffered from cardiac weakness. The feeble impulse, faint or absent heart sounds, the dilated heart, and the associated slow and irregular pulse indicated this.

Cheyne, Adams, and Stokes were careful observers, and the error into which they fell was unavoidable. Corvisart had asserted his belief that an anatomical basis for all disturbances in cardiac function could be found if one would but seek it and lamented that the study of morbid anatomy was neglected. The men of the Dublin School studied their cases anatomically as well as clinically. They were looking for alterations in the heart structure in cases of cardiac weakness and they found them. The appearance of a heart, the seat of marked fatty metamorphosis is so striking that it was but natural they should ascribe to this lesion the most serious disturbances of function.

Doubtless many factors are concerned in the production of cardiac insufficiency and probably almost every case is the result of a combination of injurious influences acting upon the heart muscle. There can be little doubt, however, that disease of the coronary arteries is one of the most frequent causes of cardiac insufficiency. And that the clinical picture which Stokes drew was not one of fatty degeneration but of arterio-sclerosis. Balfour,[51] has for many years taught that fatty heart could not be distinguished clinically.

It should be remembered that fibrous myocarditis and coronary sclerosis are not synonyms.[52] Fibrous myocarditis is rare and coronary sclerosis is common. Fibrous myocarditis although frequently associated with coronary sclerosis may occur independently. The symptoms of the two diseases are not identical, although it is often impossible to make a differential diagnosis.

If one will look over the series of cases of fatty heart collected by Stokes,[53] it will be seen that extensive disease of the coronary arteries was frequently noted. Stokes himself commented on this, but he thought coronary disease was simply an adjunct. Among the 83 cases of fatty heart collected by Quain,[54] the coronary arteries were reported ossified or obstructed in 25. In only three cases was it stated that they were healthy. Naked eye examination is not sufficient to exclude coronary sclerosis. In such cases systematic microscopical examination may reveal disease as Krehl and Dehio[55] have shown. Disease of the coronary arteries not only gives rise to the coarse anatomical alterations that have been so fully studied by Köster, Ziegler and Weigert, but to finer changes in the cells the result of interference with their nutrition and blood supply.

Sometimes examples of extreme sclerosis of the coronary arteries are found in individuals who never suffered from cardiac weakness or distress. Osler cites the case of the eminent Scotch divine Chalmers which was reported by Begbie.[56] Sudden death occurred which was attributed to advanced fatty degeneration. But the autopsy record states that "the coronary artery was loaded with calcareous deposit, much contracted, and in one place obliterated, presenting considerable resistance to the knife." I performed an autopsy for Dr. Hildreth on the body of a well-known scientist, a man of great mental and bodily vigor, who died suddenly. He had never complained of cardiac distress. The coronary arteries were found converted into calcified tubes and their lumina greatly narrowed. It is interesting to note that the arterio-sclerosis was limited to the coronary vessels. The protocol is as follows:

Anatomical Diagnosis.—*Thrombosis of right coronary artery; sclerosis of coronary arteries; chronic interstitial myocarditis; periarterial epicardial fibrous nodules; œdema of the lungs; hypertrophy and dilatation of heart; congestion of liver, kidney and spleen; chronic fibrous pleuritis.*

Body of a strongly-built and well-nourished man, aged 53 years, length 170 cm. Autopsy 22 hours post mortem. Rigor mortis present. Pupils dilated slightly, equal.

Subcutaneous fat 2 cm. thick over abdomen. Thoracic muscles deep red.

Peritoneal cavity: Omentum rich in fat. Lower border bound

[47] Welch: Medical News, 1888, LII, p. 403.

[48] Bálint: Deut. med. Woch., 1898, XXIV, p. 19.

[49] Hasenfeld und Fenyvessy: Berl. klin. Woch., 1899, Nos. 4, 6, 7.

[50] Stokes: The Diseases of the Heart and the Aorta, Philadelphia, 1854, p. 331.

[51] Balfour: Clinical Lectures on Diseases of the Heart and Aorta, Philadelphia, 1876, p. 283. Balfour: The Senile Heart, New York, 1894, p. 32.

[52] Krehl: Die Erkrankungen des Herzmuskels. Nothnagel's spec. Path. u. Ther. Vienna, 1901, pp. 313, 337. Kelle: Deutsches Arch. f. klin. Med., 1899, XLIX, p. 442. Romberg: Die Krankheiten des Herzens. Ebstein-Schwabebe's Handbuch der Praktischen Medicin. Stuttgart, 1899, p. 755.

[53] Stokes: Loc. cit.

[54] Quain: Med. Chir. Trans., London, 1850, XXXIII, p. 121.

[55] Dehio: Deutsches Arch. f. klin. Med., 1899, XXII, p. 1.

[56] Begbie: Contributions to Practical Medicine. Edinburgh, 1862, p. 196.

by light adhesions to anterior abdominal wall opposite umbilicus. Serosa pale, smooth and glistening. Appendix bound down to posterior wall near middle of course by firm adhesions. Diaphragm at 5th interspace each side.

Costal cartilages completely calcified.

Pleural cavities: Left pleural cavity free from adhesions. Right cavity completely obliterated by old fibrous adhesions, but the pleura is not noticeably thickened.

Pericardial cavity dry. Serosa smooth and glistening.

Heart weight 470 gms. There is universal hypertrophy and slight general dilatation. There is a rather thick deposit of fat over the epicardium, especially of the right ventricle. Over the coronary arteries are numerous minute pearly nodules in the substance and elevated slightly above the surface of the epicardium. Small plates similar in appearance are in the epicardium over the left coronary sinus. They average 1 mm. in size. The pulmonary artery is opened in situ and found empty. The right ventricle and auricle are distended with dark fluid blood.

The myocardium of the left ventricle contains many grayish-white semi-opaque areas of fibrous tissue, ranging in size from 0.3 cm. to 2 cm. The outlines of these islands are not sharply marked; they cut readily, the consistence not being notably greater than that of the surrounding muscle tissue, which is deep red in color. The fibrous masses are chiefly within the interior of the heart wall; a few reach the endocardium. They occur in greatest number near the middle of the ventricle, only a few are near the apex. The wall of the ventricle is of nearly uniform thickness. Wall of right ventricle normal; papillary muscles not thickened. Aortic segment of mitral valve white and opaque, but not thickened; chordæ tendineæ not shortened. Other valves normal. Root of aorta thin, elastic; intima healthy save for a few yellowish flecks.

Both coronary arteries are narrowed, thickened and largely calcified. The left, at a point 1 cm. from its origin, is converted into a calcareous tube, the lumen of which is scarcely 2 mm. in size and filled with soft yellow atheromatous material. The lumen of the descending branch of the left coronary is reduced by obliterating endarteritis to a mere slit.

In the right coronary artery, 2 cm. from origin, is a reddish-gray organizing mural thrombus 1.5 cm. long. It is firmly adherent and the narrowed lumen is plugged by a soft white mass 3 mm. long.

Measurements: Tricuspid valve 14 cm.; pulmonary valve 7.5 cm.; mitral valve 10.5 cm.; aortic valve 9 cm.; left ventricle 1.6 cm. thick; right ventricle 0.4 to 0.5 cm. thick.

The lungs are voluminous, moderately œdematous and congested. They are crepitant throughout.

Spleen weight 170 gms. Rather firm; four notches in anterior border. It cuts readily, and is deep red on section; pulp not increased.

Stomach and intestines appear normal.

Liver does not extend below costal margin. Normal consistence; lobular markings rather obscure. Gall bladder normal.

Kidneys: Equal size. One measured 12 x 5.5 x 4 cm. and weighed 200 gms. Firm, cuts easily. Capsule strips readily from a smooth dark red surface. On section dark red. Cortex 8 mm. wide; glomeruli appear as deep red points. Neither branches of renal arteries nor main artery notably thickened.

Aorta smooth, wall not thickened, elastic; few areas of fatty degeneration in intima.

Brain weight 1400 gms. Both hemispheres symmetrical. Pia normal. Vessels at base not sclerotic.

Microscopic examination of fresh tissues: No fat visible in kidney or heart muscle.

Microscopic examination.—Heart: The white firm areas detected on naked-eye examination are composed of rather loose areolar connective tissue containing thin-walled blood-vessels filled with blood. From these areas the muscle cells have entirely disappeared. Elsewhere the muscle fibres, save for slight vacuolization, appear normal. There is no diffuse general increase of the interstitial connective tissue. The smaller arteries are apparently not thickened or diseased.

Right coronary artery, 2 cm. below origin: The wall is almost completely converted into hyaline fibrous tissue. The media is thinned, except in a small portion of the circumference. Here only the internal limiting membrane is well preserved. The projection into the lumen, noted at the autopsy, is due to a localized thickening of the intima and is composed of homogeneous hyaline connective tissue, containing numerous slits running transversely. The surface of this nodule is smooth.

Left coronary near origin. The hyaline degeneration is even more marked than in the right coronary. The media is greatly thinned and degenerated. A thick broad layer of the thickened intima has been split off and one end projects into the middle of the lumen. The free end is surrounded by blue-staining amorphous material (atheromatous debris), and beyond this is loose areolar fibrous tissue containing thin-walled vessels. In the meshes are numerous large cells filled with yellow pigment (organization of a blood clot?). A band of this tissue extends from wall to wall through the centre of the lumen. In addition to the thick band, bits of the intima have been torn loose at several points.

Spleen: Hyperæmic; the pulp is filled with red blood corpuscles so that its framework is obscured.

Liver: The intralobular capillaries are uniformly dilated. No sclerosis of blood-vessels.

Kidney: Congested.

It is probable as F. H. Pratt[87] has pointed out that in instances of slow obliteration of the arteries the coronary venous channels especially the veins of Thebesius aid the scanty arterial blood supply sufficiently to maintain the nourishment of the muscle fibres.

The notable progress that has been made in the study of cardiac insufficiency during the last dozen years is largely due to the work of the Leipsic school, especially in the researches of Krehl and Romberg. The work of this group of investigators has emphasized the relation which exists between disturbances of cardiac function and demonstrable lesions in the heart muscle. Krehl[88] found areas of acute interstitial myocarditis in every case of chronic valvular disease examined, and also in the so-called idiopathic cardiac hypertrophy. Romberg[89] and Kelle[90] demonstrated the great frequency of foci of interstitial myocarditis in acute infectious diseases. E. Albrecht[91] has examined twenty-one hearts by Krehl's method and confirms the findings of the Leipsic investigators. Dehio of Dorpat has described a diffuse myofibrosis of the heart occurring frequently in old people. Dehio[92] thinks this formation of fibrous tissue is a compensatory process and regards it as an attempt to strengthen the weak heart walls analogous to Thoma's theory of the forma-

[87] F. H. Pratt: Am. Journ. of Phys., 1898, 1, p. 86.

[88] Krehl: Deut. Arch. f. klin. Med., 1890, XLVI, p. 454. Ibid., 1891, XLVIII, p. 414.

[89] Romberg: Ibid., 1891, XLVIII, p. 369. Ibid., 1892, XLIX, p. 413.

[90] Kelle: Ibid., 1892, XLIX, p. 442.

[91] E. Albrecht: Der Herzmuskel. Berlin, 1903, p. 200.

[92] Dehio: Deut. Arch. f. klin. Med., 1899, XXII, p. 1.

tion of connective tissue in the walls of weakened vessels in arterio-sclerosis. This myofibrosis is often strongly marked in the auricles and may cause serious disturbance from the fact that the stimulation which leads to the systole originates in the auricles near the great veins.

The results of these investigators are based upon the most careful and laborious examination. Krehl's method, which was used also at Dorpat in a modified form, consists in cutting up the entire heart into blocks 1 cm. to 1½ cm. thick and then examining a section from each block.

It must be admitted, however, that all cases of muscle incompetence can not be explained on the basis of definite anatomical changes. Rieder in Bollinger's laboratory has shown that as a rule in so-called idiopathic cardiac hypertrophy, sufficient anatomical alterations are not present.

Fatty overgrowth of the heart or obesity of the heart as older writers termed it, is a feature of general corpulency. There is associated with deposits of fat elsewhere, an excessive accumulation beneath the pericardium, and between the muscle fibres. The adipose tissue may be present in enormous amounts, without disturbing the function of the heart as in a case reported by v. Leube.

The size of the heart is proportionate to the size of the skeletal muscles but the work of the heart is proportionate to the bulk of the body. Hence in obesity an increased amount of work is thrown on the heart. Corpulent individuals frequently have very small and weak muscles and hence the disproportion between the size of the heart and the work to be done is increased. Traube noted many years ago the difference in the behavior of the heart in stout people with strong muscles and stout people with weak muscles.

Cardiac insufficiency may be due to acute over-distension of the heart produced by sudden and violent bodily exertion. In these cases of heart strain the nature of the anatomical or chemical changes is unknown.

No justification exists for attributing cardiac insufficiency to nervous disturbances or exhaustion, the nature of which we know nothing, unless demonstrable lesions in the myocardium have been excluded by a thorough microscopic examination. It should be stated that the results of recent work indicate that the circulatory disturbances which occur during the height of the febrile period in the infectious diseases are probably due less to cardiac insufficiency than to paralysis of the vaso-motor centre in the medulla.[a] The blood pressure falls, the flow of blood is slowed, the heart is only partially filled, and finally the circulation sinks to such a low level that life cannot be maintained.

It is probable that many of the problems connected with cardiac insufficiency can be solved by careful clinical and pathological study. The advances made by the Leipsic school were due to a thorough study of a few cases. There is great need of additional data.

In conclusion it may be stated that there is no evidence to show that fatty metamorphosis of the heart produces cardiac insufficiency. In the light of present knowledge other anatomical alterations, especially coronary sclerosis, and acute interstitial myocarditis must be regarded as the most common causes of heart failure.

[a] Romberg und Pässler: Deutsches Arch. f. klin. Med., 1899, LXIV, p. 652. Pässler und Rolly: Münch. med. Woch., 1902, XLIX, p. 175. Pässler und Rolly: Deutsches Arch. f. klin. Med., 1903, LXXVII, p. 96.

NOTES ON FORM OF THE CAVITY OF THE KNEE-JOINT.

By Joseph Marshall Flint, M. D.

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It has been a well-known psychological fact that the sensorium finds it difficult to apprehend ideas of positive form when expressed in negative outlines. This has been repeatedly shown by experiment, and is a fact which must have impressed every student of anatomy in the endeavor to obtain clear conceptions of the form of the various cavities in the body. This is particularly true of the pneumatic sinuses of the cranium and of the form of the pleural and peritoneal spaces, as well as the various cavities of the joints. Recently Lee[1] has emphasized this point in a study from this laboratory on the form of the sinus frontalis as displayed by lead casts obtained in the dissecting-room during the periods of routine work. By this method he has succeeded in giving us very clear and definite objective pictures of these negative cavities. When a similar method is adopted for the study of such intricate spaces as the synovial cavity of the knee-joint, many interesting relationships of the different porfions of the cavity to each other as well as to the bones that form its boundary are revealed. These cavities, in their normal form, have been the subject of considerable minute study by numerous anatomists. The casts yield nothing new so far as the joint cavity itself is concerned, but give us in three dimensions an excellent objective concept of the form of this space. A partial idea of the outlines of the synovial sac has been obtained by injecting the cavity with gelatine or wax under considerable pressure[2]; but the picture ob-

[1] Lee: Johns Hopkins Hospital Bulletin, April, 1904.

[2] Vide: Poirier et Charpy. Traité d'Anatomie Humaine. T. 1. Paris, 1899. Spalteholz: Handatlas der Anatomie des Menschen. Leipzig, 1896. Fick: Handbuch der Anatomie des Menschen. Jena, 1904.

tained in this was is only partial, inasmuch as all the relationships of the space to the condyles of the tibia and femur respectively are hidden, as well as the extensions of the cavity above and below the semilunar cartilages. In demonstrating the form of this space to students, it has been my practice to inject the cavity with molten Wood's metal, which has a melting-point at about 63° C. The technique is extremely simple. After the joint has been freed from muscles and ligaments, it is immersed in a pan of water having a temperature of about 65°, and is left until the whole specimen is warmed through. After this has taken place, a small hole is made in the capsule and molten Wood's metal poured into the cavity of the joint. The joint is then placed in any desired position, and plunged into cold water, whereupon the metal immediately hardens, and can be easily dissected out, yielding a complete picture in positive form of the shape of the joint cavity. Under ordinary circumstances, it is extremely difficult to inject the entire space at once, and in consequence it is best to make casts from above and below by opening the cavity at the upper extremity of the subpatellar bursa and through the popliteal space. The latter shows the relationship of the extension of the cavity beneath the patella and tendon of the quadriceps femoris while the former demonstrates particularly well the synovial space between the femur and tibia. By superimposing two such casts, one obtains by the combined picture a perfect representation of the cavity at its maximum distention. They serve admirably for the purposes of demonstration, or for museum specimens, and, at the same time, afford excellent material for illustration. Owing, of course, to the fact that the muscular tonus has been removed, as well as any elasticity that the capsule of the joint itself might retain, the form of the space as shown by these specimens must be considered maximal, and paralleling the conditions occurring in life only when we obtain a large effusion into the cavum articulare.

Literature:

The descriptions of the synovial cavity of the knee-joint as given by the more thorough and standard works on anatomy agree in the main upon the chief characteristics and relationships of the synovial membrane to the ligaments, capsule, cartilages and bones entering into the formation of the joint. As for the general descriptions of the negative spaces, these leave little to be desired. A brief reference to one or two of them accordingly will be sufficient to review our knowledge of these spaces as obtained by the older methods. It would be extremely desirable, however, to have better studies of the average form of this and indeed of all synovial cavities, such as might be obtained from a large number of casts. They could be made not only in full extension like the ones used in this paper but also to show the effect of the various movements upon the form of the cavity. Naturally the difficulty lies chiefly in the expense involved, for Wood's metal of which the casts are made is costly.

According to Morris,[3] the synovial membrane of the knee-joint limits the largest synovial sac in the body. Following the capsule of the joint, it opens into a large cul de sac beneath the extensor tendon on the front of the femur, and communicates with the large bursa interposed between the tendon and the femur. After investing the circumference of the lower end of the femur, it is reflected upon the capsule of the joint which forms the anterior, posterior and lateral ligaments. The crucial ligaments are also covered. The membrane extends over both surfaces of the semilunar cartilages and thence along the inner surface of the coronary ligaments to the head of the tibia, around the circumference of which it extends a short way, and sends an extension to form a bursa for the popliteus tendon. At the back of the articulation two pouches are prolonged beneath the muscles, one on either side, between the femur and head of the gastrocnemius. Another invagination, less sharply differentiated, occurs around the patella. From it arise the alar ligaments.

Spalteholz[4] in a brief description states that the cavum articulare of the knee-joint is regularly in open connection with the bursæ of the popliteus muscle This tubular projection appears on the lateral side, and runs from the under surface of the tendon of origin of the popliteus downwards and backwards. The communication takes place through a small slit above the meniscus lateralis. Beneath the meniscus there is sometimes a second communication, joining the cavum articulare with the tibio-fibular articulation. Above, the cavum is in communication with the bursa suprapatellaris, which lies between the femur and the common tendon of the M. quadriceps femoris. This is customarily cut or traversed by folds, and may at times remain completely separated from the remainder of the knee-joint.

Cunningham[5] described the joint cavity, or rather the synovial membrane which forms its boundary, as a more or less extensive lining of the capsule and the intracapsular ligaments and the free surface of the infrapatellar pad of fat. Upon its surface the membrane forms a fold extending from the lower level of the articular surface of the patella to the anterior portion of the intercondyloid notch. It then extends upward upon the top surface of the extensor tendon, communicating with a large bursa on the front of the femur. Below it covers both surfaces of the semilunar fibro-cartilages, while a prolongation invests the intracapsular portion of the tendon of the M. popliteus. From the back portion of the joint cavity the synovial membrane extends upward, and provides a partial covering for the crucial ligaments. The cavity may, furthermore, communicate with bursæ situated on the inner head of the gastrocnemius and the semimembranosus muscles. Occasionally the communication occurs between the cavities of the knee-joint and that of the superior tibio-fibular articulation.

Cavum articulare:

Casts to display the region between the femur and the patella are best made from an opening in the popliteal space.

[3] Morris: Anatomy of the Joints. London, 1879.

[4] Spalteholz: Loc. cit.

[5] Cunningham: Text Book of Anatomy. New York, 1902.

In almost all instances the suprapatellar bursa (Figs. 1, H, 2, a, 3, c, 4 a, 6, 1.), communicates with the joint cavities. So regular is this connection that it may readily be considered and described with the cavum articulare. The size and relations of this projection or diverticulum of the synovial cavity vary within comparatively wide limits in both form and relationship. Commonly it is cut off by a septum (Figs. 1, a, 4, h, 6, a.), from the lateral side which projects a considerable distance across the space, and, in rare instances, entirely separates the bursa from the joint. The

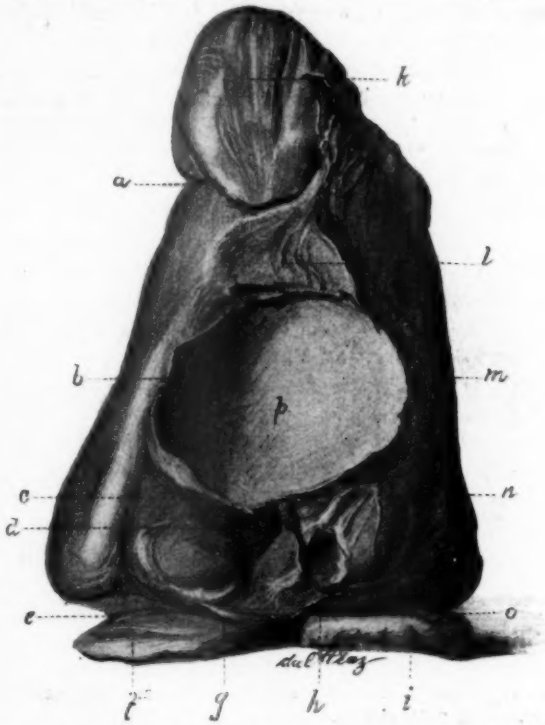


Fig. 1.—Wood's metal cast of the cavum articulare of the right knee-joint. Anterior aspect. ¾ natural size.

a=Septum between bursa suprapatellaris and the cavum articulare.
b=Peripatellar ridge formed by a synovial groove aronnd the patella.
c=Plica aliformis lateralis.
d=Foramen for ligamentum mucosum passing from lower border of patella to intercondyloid notch.
e=Space for meniscus lateralis.
f=Extension of cavum articulare between the meniscus lateralis and the facies articularis of the condylus lateralis of the tibia.
g=Groove for the so-called ligamentum anterius of the meniscus lateralis.
h=Notch for ligamentum cruciatum anterius.
i=Extension of cavum articulare between the meniscus medialis and the facies superior of the condylus medialis.
o=Space for the meniscus medialis.
n=Plica aliformis medialis which, with its mate c., forms the plicae alares.
m=Medial ridge formed by the peripatellar groove.

communication between the two sacs is almost always on the medial and posterior side. This bursa may extend a distance of 6 or 7 cm. above the upper level of the patella. Ordinarily it is rounded, and, when distended to a maximum, appears to be between 3 and 4 cm. in thickness. Upon its surface, as shown by the casts, is a distinct depression for the tendon of the M. quadriceps femoris. In certain cases, the bursa may appear as a direct continuation of the synovial sac, but from its upper extremity distinct diverticulæ (Figs. 2, c, 3, a.), may extend out to increase the area and size of the bursa. At the same time lateral and longitudinal folds of the synovial membrane frequently form depressions and sulci upon the surface of the casts. These appear most numerous on the surface of the bursa suprapatellaris and the point of reflection of the membrane from the sides of the condyles of the femur.

Anterior View Upper Part of Cavum Articulare Showing Diverticulum from the Bursa.

Fig. 2.—Wood's metal cast of the upper part of the cavum articulare of left knee-joint and the bursa suprapatellaris showing a diverticulum from the bursa. Anterior view. ¾ natural size.

a=Bursa suprapatellaris.
b=Impression of patella with lateral and medial facies. Above the patella the impression of the tendon of the M. quadriceps femoris is visible.
c=Diverticulum.

The bursa suprapatellaris commonly extends a distance of from 2 to 3 cm. above the upper edge of the articular surface of the femur. The cavum articulare, as shown by the cast viewed from above and behind (Fig. 3), is divided at the level of the intercondyloid notch, where there is always a for-

Posterior View of Same Cast Shown in Fig. 2. ¾ Natural Size.

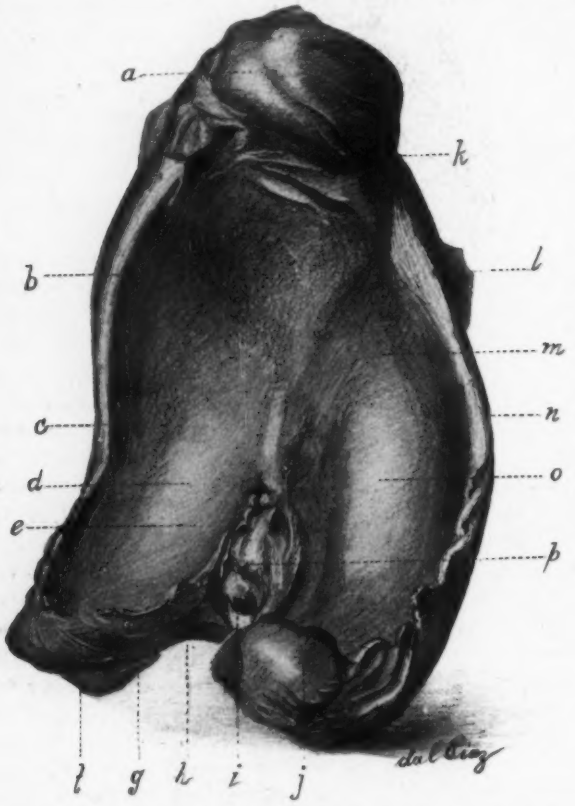
Fig. 3.—Lateral and posterior view of same cast as shown in Fig. 2.

a=Diverticulum from suprapatellar bursa.
b=Septum or fold of synovial membrane in lateral wall of cavum articulare.
c=Bursa suprapatellaris.
d=Impression of the condyles of the femur.

amen made by the ligamentum mucosum, as it passes from the alar ligaments of the patella to its insertion upon the upper part of the intercondyloid notch. Above this point, the impressions of the femoral condyles are clearly seen as well as the mark of the facies patellaris femoris. From this

medial point, the cast not only shows very clearly the extension posteriorly of both condyles but also the projection of the sac into the intercondyloid fossa as well as the line of its reflection all the way around the condyles of the femur. At the edge of the femoral condyles about the line of attachment of the capsule, there may be a considerable thickening of the cast, indicating that the synovial sac is large at these points. Ordinarily this fold is thicker on the external than on the internal condyloid edge of the femur. Viewed from behind and above, the cast also shows a very marked notch (Fig. 4), between the two condyles where the synovial membrane is reflected or passes over the crucial ligaments. The relationship of the membrane to the ligamentum cruciatum anterius is clearly shown, as this forms by its partial synovial investiture the major portion of the notch. Anteriorly the continuation of the ligamentum cruciatum anterius with the meniscus medialis is distinctly seen as a definite groove. Above, the limits of the condyle are very evident; but there is, however, no indication of the presence of the patella, as that, on account of the distention of the cavum articulare, is pressed some 2 cm. away from the patellar surface of the femur.

The anterior aspect of the synovial cavity (Fig. 1) is limited above by the bursa suprapatellaris and the extensor tendon (Fig. 1, k and 1), in the middle, by the articular surface of the patella (Fig. 1, p), and the capsule of the joint, and below by the two semilunar cartilages and coronary ligaments (Fig. 1, e, o, f and i). In general, the entire form of the cast is pyramidal, although quite irregularly so. Above, the rounded apex is formed by the bursa suprapatellaris, which we have previously described. Following the attachment of the capsule and the fibrous expansions of the extensor tendons immediately above the patella is the sulcus in the tendon itself. The patella is shown as a deep depression in the cast, its medial and lateral facets clearly visible.



POSTERIOR VIEW OF CAVUM ARTICULARE OF KNEE-JOINT. SAME CAST AS FIG. 1. ¾ NATURAL SIZE.

FIG. 4.—Posterior view of same cast of the cavum articulare as shown in Fig. 1.

a=Bursa suprapatellaris.
c=Margo medialis of the cavum articulare marking the line of reflection of the synovial membrane from the femoral condyles to the capsule of the joint.
b=Superior margin of the condylus medialis.
d=Impression of the condylus medialis.
e=Foramen for the transmission of the ligamentum mucosum.
f=Portion of cavum articulare between the meniscus medialis and the posterior part of the condylus medialis of the femur.
g=Extension of the synovial cavity between the meniscus medialis and the superior face of the condylus medialis of the tibia.
h=Groove for the ligamentum cruciatum posterius.
i=Groove for the ligamentum cruciatum anterius.
j=Portion of cavum articulare at the posterior surface of the condylus lateralis of the femur.
k=Septum between the bursa suprapatellaris and the cavum articulare.
l=Ridge about the patella.
m=Upper margin of condylus lateralis of femur.
n=Lateral margin of the cavum articulare where the synovial membrane is reflected from the lateral condyle to the capsule of the joint.
o=Impression of the condylus lateralis.
p=Extension of the cavum articulare behind the origin of the lig. cruciatum anterius in the intercondyloid notch.

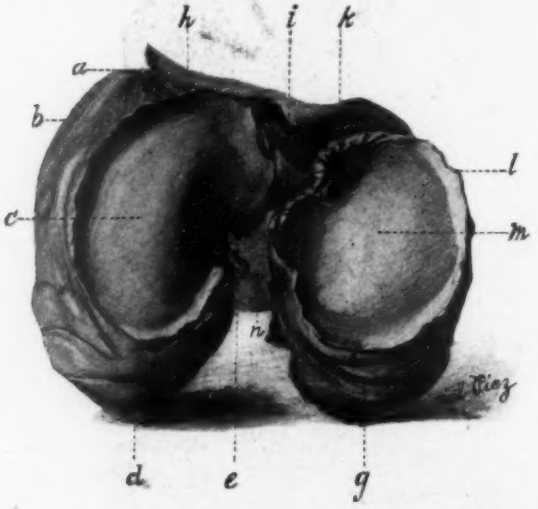


FIG. 5.—Under surface of the cavum articulare from same cast as Fig. 1. ¾ natural size.

a=Depression about patella formed by extension upwards of the plica aliformis lateralis.
b=Lateral limit of portion of cavum articulare between the meniscus lateralis and the condylus lateralis of the tibia.
c=Impression of the condylus lateralis of the tibia.
d=Portion of cavum articulare between the condylus lateralis of the femur and the capsula articularis.
e=Groove for the ligamentum cruciatum posterius.
g=Portion of cavum articulare between the condylus medialis of the femur and the capsula articularis.
h=Lateral articular facet of the patella.
i=Groove for the ligamentum cruciatum anterius.
k=Medial articular facet of the patella.
l=Medial limit of the portion of the cavum articulare extending between the meniscus medialis and the condylus medialis of the tibia.
m=Impression of the condylus medialis of the tibia.
n=Bursa suprapatellaris seen through the notch formed by the crucial ligaments.

It is surrounded by a sharp, elevated ridge formed by the infolding or invagination of the synovial membrane around the edge of the patella which may be termed the peripatellar groove (Fig. 1, m and b). Immediately below is a depression which terminates in a foramen caused by the ligamentum mucosum as it passes from the patella to the edge of the intercondyloid notch. The depression which converges from the borders of the patella is formed by the plicæ alares (Fig. 1, c, n). Below the foramen, we find a notch in the cast (Fig. 1, h), formed by the ligamentum cruciatum anterius as it passes forward, and it is attached into the fossa in front of the spine of the tibia just behind the transverse ligament. This notch is continuous with the depression formed by the

Fig. 6.—Lateral view of the Cavum Articulare of the knee-joint. Same cast as Fig. 1.

a=Septum between the bursa suprapatellaris and the cavum articulare.
b=Impression of the condylus medialis of the femur.
c=Groove for ligamentum cruciatum anterius.
d=Projection of the cavum articulare into the intercondyloid notch behind the origin of the ligamentum cruciatum anterius.
e=Portion of the cavum articulare between the condylus lateralis of the femur and the capsula articularis. The folding is due to reduplications in the capsule of the joint.
f=Space occupied by the meniscus lateralis.
g=Extension of the cavum articulare between the meniscus lateralis and the condylus lateralis of the tibia.
h=Bursa suprapatellaris.
i=Impression of the patella.
k=Groove about the patella.
l=Depression formed by the plicæ alares and the ligamentum mucosum.
m=Lateral margin of the cavum articulare.

meniscus medialis. On the opposite side is a well marked groove (Fig. 1, g), formed by the ligamentum transversum genu, which is continuous with the depression (Fig. 1, e) or the anterior and medial aspect of the depression formed by the meniscus lateralis. The extensions of the cavum articulare beneath the two semilunar cartilages are very beautifully shown, extending out to the margin of the condyles of the tibia where they are limited by the so-called coronary ligaments.

The cast of cavity of the knee-joint, viewed from below or better from the tibial aspect (Fig. 5), shows the articular surfaces of the two condyles of the tibia represented as concave surfaces with the thin, sharp edges marking the line of attachment between the capsule of the joint and the condyles of the tibia. The synovial space between the condyles and the semilunar cartilages is extremely thin and delicate, and extends ordinarily only over the anterior three-fourths of the condylar surface. Here in the tibial as well as in the femoral view, the distinctly marked notch formed by the reflection of the synovial membrane over the crucial ligaments is shown (Fig. 5, n). In addition, there is a secondary notch where the anterior crucial ligament passes to the point of its attachment near the anterior border of the meniscus medialis (Fig. 5, i). A synovial diverticulum passes in front of the crucial ligament at this point, and is derived from the part of the cavity between the condylus medialis and the meniscus medialis. A depression at the lateral edge of the intercondyloid notch of the femur shows the impression of the posterior crucial ligament (Fig. 5, e). The lateral aspect of the cast (Fig. 6) shows, particularly at the point of attachment of the capsule to the lateral condyle of the femur, numerous rugæ (Fig. 6, e), caused by the folding of the synovial membrane at this point. Above, the cast becomes more rounded, and finally opens into the suprapatellar bursa (Fig. 6, h). The projection about the edge of the patella (Fig. 5, h), forming the peripatellar groove is distinctly and clearly marked. The medial aspect is somewhat flatter and sharper than the lateral, and does not show the same marked folding as is observed on the lateral aspect of the cast. Within, the impressions of the femoral condyles are seen while at the intercondyloid fossa just in front of the notch formed by the crucial ligaments (Fig. 6, c), is a slight projection of the cavum articulare into the intercondyloid fossa. Behind the notch is another projection (Fig. 6, d), extending into the fossa posterior to the ligamentum cruciatum anterior.

In general, the form varies considerably in different joints, and the complicated infoldings and relationships shown by these specimens indicates only too well the difficulty in trying to get an adequate concept of the form of the cavum articulare from ordinary dissections or even from the distended synovia which can be obtained by injecting the cavity with gelatine or other substances. It emphasizes, moreover, the necessity of positive expressions of these negative spaces in order to give direct and visual images which we can project into the cavity of the joint itself. The surgical interest of these casts, moreover, is of not a little importance, as in extensive effusion, either purulent or serous, into the cavum articulare must necessarily take the form which is shown by

these casts. The reason why, for example, effusions into the joint are never visible or palpable from the popliteal space becomes immediately apparent, for in this region the extensions of the synovial cavity are not marked. They also indicate the areas where an opening into the joint would give access to the greater part of the cavity, and suggest also the regions which should be carefully observed in washing out or aspirating the joint in various surgical procedures.

THE REPORT OF A CASE OF ACUTE MYELOGENOUS LEUKÆMIA.

By Hermon C. Gordinier, M. D., Troy, N. Y.

The only form of acute leukæmia mentioned by the authors of our modern text-books of practice is the lymphatic. No mention so far as I have been able to ascertain is made of the acute myelogenous form. Of the various works on hæmatology at my disposal only three, Ewing, Grawitz, and Bramwell mention this type of leukæmia.

Ewing states "That, of the recent cases of acute leukæmia in which the type of the disease was satisfactorily determined, Fraenkel (1895), could find only three of the myelogenous type, the great majority being of the lymphatic variety.

In 1897, the writer saw three cases of rapidly fatal leukæmia verified by autopsies, and all of the myelogenous type. They occurred in the services of Drs. Thompson and Delafield at Roosevelt Hospital. In two of the cases the changes in the blood were from the first observation typical of the condition and the marrow was puriform, while the spleen and lymph nodes were but slightly affected. In the third case on the first examination there was found a leucocytosis of ordinary inflammatory grade, five per cent of the cells were, with difficulty, recognized as myelocytes. Later the leucocytosis and the proportion of myelocytes increased and the diagnosis of acute myelogenous leukæmia, made with reserve, just before death, was verified at the autopsy."

Grawitz, reports the case of a spinster, age forty-two years, with previous good health. This condition followed a long continued hemorrhage, the result of extracting fourteen teeth at one sitting. The patient had a severe stomatitis and necrosis of the jaw; this was followed by great prostration, retinal hemorrhage and enlargement of the spleen. The diagnosis of acute leukæmia was made. Leucocytes 190,000; small mononuclears five per cent, large mononuclears fifteen per cent, polynuclears ten per cent, sixty per cent neutrophile myelocytes and ten per cent eosinophile myelocytes. Death occurred in two weeks.

Bramwell in his work on anæmia mentions acute myelogenous leukæmia and refers to the following case with recovery. "A labourer came under my care in 1870. He had been ill three weeks, there was some, though not marked, febrile disturbance. The patient was pale and cachectic looking, his color resembled that of pernicious anæmia, the spleen extended down almost to the pelvis, the lymphatic glands were not enlarged, there was no hemorrhage, the blood contained an enormous excess of white cells, they appeared quite as numerous as the reds. At that time physicians were not in the habit of counting the corpuscles, and I cannot say what the exact proportion of the white and red cells was, the appearance of the blood was in every way characteristic. I was well acquainted with the disease, splenic leucocythæmia as it was then termed from Bennett's teaching and from the cases which I had seen in the Edinburgh Royal Infirmary. The case just recorded is so far as I know unique, inasmuch as it ended in recovery, and that, so far as I could judge from the microscopic characters of the unstained blood, the white cells were of large size and coarsely granulated, in other words the case was one of acute spleno-medullary (and not lymphatic) leucocythæmia."

In the American Journal of the Medical Sciences for September, 1903, Billings and Capp in an exhaustive article directs the profession's attention to acute myelogenous leukæmia. They reported one case and collected from the literature seven other positive cases, thus establishing this type of leukæmia as a distinct clinical entity. Heretofore the only form of acute leukæmia generally recognized was the lymphatic. Since the appearance of their article I have been able to collect from the literature, ten cases of acute leukæmia all of which were of the lymphatic type. I also found an undoubted case of acute myelogenous leukæmia reported in 1902, by Stewart and Campbell. (This case seems to have been overlooked by Billings and Capp). It was in a child six years of age, the blood showed, toward the end of the disease which ran its course in two months, the following condition. Hæmoglobin twenty-one per cent, Red cells 750,000, leucocytes 40,000; lymphocytes small twenty-five per cent, myelocytes thirty per cent.

Author's Case.

A. C. T. Age fifty-six years, married, merchant by occupation. Admitted to my service at the Samaritan Hospital, April 4, 1903, complaining of great weakness, cough, sleeplessness, and complete anorexia.

Family History.—Mother died of bronchitis. Father of small-pox. Lost one brother of tuberculosis. Has a brother and sister living and in good health.

Personal History.—Fourteen years ago had a severe attack of *lagrippe*, otherwise has been healthy until onset of present illness. Has used tobacco to excess, but never used alcoholics in any form. Denies venereal disease of any sort. Present illness began toward the end of December, 1902, with a severe attack of bronchitis which was followed in the course of a few weeks by great fatigue, loss of appetite,

chilly feelings, fever and sweats, constant headache, dyspnœa on exertion, throbbing in the head and insomnia. He thinks he has lost about thirty pounds in weight. His cough continues, and he expectorates a muco-purulent sputum.

Physical Examination.—The patient assumes the recumbent posture being too ill to walk. Is of large frame, well built, but with flabby musculature. There is slight general emaciation. Has a cachectic look, color of face of a dusky yellow hue; lips, conjunctivæ and all visible mucous surfaces very pale, gums spongy and bleed easily when rubbed, tongue large, flabby and coated with a white fur. A superficial ulceration exists on left side of tongue near its tip, the size of a silver three cent piece. No cyanosis, no oedema of extremities, superficial veins distended.

Lungs.—Chest symmetrical, intercostal spaces widened, respirations hurried. Percussion note universally vesiculo-tympanitic. The upper liver dulness and the cardiac areas are effaced by this vesiculo-tympanitic note. Expiration prolonged and low in pitch, a few dry and moist râles are heard.

Heart.—The pulse is regular but rapid (120-140 per minute), easily compressed and of fair volume. The apex is not visible or palpable, the muscular element of the first sound is weak, a slight apical systolic murmur is present, not conducted to the left. The pulmonic and aortic sounds retain their normal disparity. A distinct venous hum is present on each side.

Liver.—The hepatic dulness reaches, in the mammillary line from the sixth rib to three centimeters below the free border. The dulness of the left lobe extends six centimeters below the zyphoid cartilage. The lower liver border is palpable, firm in consistency, not irregular, and slightly tender on pressure.

Spleen.—This organ is palpable by dipping one's hand deep below the costal border. Its dullness begins in the eighth interspace and extends downward, in the mid-axillary line, eleven centimeters. Forcible percussion of the spleen elicits no tenderness.

The abdomen is distended, not tender, and generally tympanitic. No evidence of free fluid exists.

Glandular System.—A solitary lymph node is present in the left anterior triangle of the neck, a few pin-head sized lymph nodes exist in each groin, none others are detectable.

Urine.—Light amber, specific gravity 1022, no sugar, trace of albumen, no blood, pus or casts, amorphous urates and abundance of uric acid crystals. Diazo-reaction absent. Amount three pints.

The sputum which was examined several times, disclosed diplococci.

Nervous System.—Mind clear to last. Muscular movements normal but weak. No ataxia, sensations intact. Sternum, ribs and long bones of extremities tender when struck. Pupillary reactions normal. Ocular movements normal. Flamed shaped hemorrhages in each retina. Hearing reduced to three inches from each ear, drum membranes thickened and retracted. The knee-jerks are diminished, otherwise the reflexes are normal. No nerve or muscular tenderness.

Blood.—The freshly drawn blood is thin, runs easily and coagulates slowly, and has a slight whitish tinge. Hæmoglobin twenty-eight per cent (Dare, Tallquist). Erythrocytes 1,500,000 per cmm. Leucocytes 260,000 per cmm. The blood presents a typical picture of myelogenous leukæmia, the specimens, stained with Wright's modification of the Leishman stain, showed a well marked mixed leucocytosis, the myelocytes predominating. A slight degree of poikilocytosis exists. The protoplasm also showed a mild degree of polychromatophilia, but no granular degeneration. Nucleated red cells were common, chiefly of the normoblastic type. An occasional megaloblast was present, perhaps one to every second or third field. No count of the nucleated red cells was made. Numerous eosinophile myelocytes were present containing characteristic coarse and fine granules and pale stained eccentrically placed nuclei. It was often difficult to distinguish between some of the large mononuclear leucocytes and myelocytes with faintly stained granules. Only a few polymorphonuclear eosinophiles were present, an occasional dwarf form was observed. A few mast cells with characteristic basophilic granules were seen. The blood contained many spheric bodies smaller than the average red cell, without a distinguishable surrounding seam of protoplasm. At first, these were thought to be a small type of lymphocyte, but after a more careful study they were considered to be free nuclei which had taken the blue stain. A few myelocytes exhibited mitotic figures. Many degenerated types of leucocytes existed.

Blood Count.	Per cent.
Myelocytes, Neutrophilic	61.4
Myelocytes, Eosinophilic	4.1
Transitionals	1.8
Polynuclear Eosinophiles	.2
Polynuclears	5.8
Mononuclears, Large	24.4
Mononuclears, Small	2.
Mast Cells	.3
	100.

The patient died April 9, 1903, five days after entrance to hospital. No post-mortem examination was permitted. There can, however, be no question about classifying this case as one of acute myelogenous leukæmia. The duration of the symptoms referable to the blood-state was a little over three months (from about January 1, 1903, to April 9, 1903), and the blood picture was absolutely typical of myelogenous leukæmia.

This case together with those recorded in literature brings the number of acute cases of myelogenous leukæmia up to twelve positive and two doubtful ones. The positive cases are recorded by Billings and Capp; (1) Vander Wey; (2) Thompson and Ewing; (3) Grawitz; (4) Ewing; (5)

Hirschfeld and Alexander; (6) Michaelis; (7) Bramwell; (8) Stewart and Campbell; (9) Turck; (10). The two doubtful cases are recorded by Obrastow; (11).

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THE HISTORY OF SOME FAMOUS QUACKS.[1]

BY FRANCIS R. PACKARD, M. D., of Philadelphia.

Although a dissertation on successful quackery may seem out of place before an audience of honest physicians, nevertheless, there have been, and unfortunately still are some phases of the subject which may claim attention because of their human interest, although they are destitute of scientific value. There is in most successful quacks some distinctive quality or characteristic to account for their achievements. Paracelsus, who may serve as the prototype of the modern quack, was undoubtedly a man of intellectual attainments, and possessed of supreme cleverness. He also possessed a characteristic, which seems to be an essential requisite to all successful quackery, namely the most superb self-confidence. His very name is indicative of his career,—Aurelius Phillippus Theophrastus Paracelsus Bombastus ab Hohenheim, and savors of his magnificent pretensions. It is probably impossible for us to appreciate the astounding audacity of his action when, while professor of Physic and Surgery, in the University of Basel, he burnt the works of Galen and Avicenna publicly in his lecture room, accompanying the performance by a speech in which he modestly claimed that all the academies had less experience than his beard, and that his hat knew more than all the doctors. In spite of Browning's wonderful poem, which may be regarded as an attempt at an apotheosis of Paracelsus, there is no doubt that he was a quack pure and simple. His sole credit was that he broke from the traditions of the past and shattered some of the idols of medieval medicine.

Webster defines a quack as "a boastful pretender to medical skill; an empiric, an ignorant practitioner." Those to whose careers I wish to call your attention briefly to-night belong to the first class. They were all boastful pretenders to medical skill, though some of them were not so ignorant. Under this head for many generations we might have classed the sovereigns of England, the mere touch of whose hand was held to be an absolute cure for scrofula, a therapeutic measure from which the condition derived its other name, "King's Evil." That this opinion was not confined alone to the uneducated classes was demonstrated by the fact that the august possessors of the virtue not only practised it but repeatedly asserted their claim to it.

During the period of the restoration especially submission to the royal touch as a cure became very popular as a manifestation of loyalty. Another circumstance which contributed to maintain the royal touch in popular estimation was the fact that it was always accompanied by the gift of a gold piece to the patient from his sovereign. These were known as "touchpieces." It is interesting in this connection to recall that Dr. Samuel Johnson, the great lexicographer, was among those touched for this complaint by Queen Anne, the last of England's royalties to attempt the exercise of this healing faculty.

VALENTINE GREATRAKES.

In twenty-two years a careful list was kept of those of his subjects whom Charles II touched for King's Evil, and their number amounted to over 92,000. There were, however, other claimants to the healing touch besides those of royal birth, among whom Valentine Greatrakes, achieved a remarkable fame. He was born in County Waterford in the reign of Charles I, and after an ordinary school education entered Trinity College, Dublin, from which place he did not graduate as, owing to the rebellion his mother fled from Ireland into England, where he lived in Devonshire and studied theology and philosophy under a German minister, the Reverend John Daniel Getsius. About the year 1624, he returned to Ireland, but he is said to have been so extremely affected by the miserable state of that unfortunate country that he retired to the Castle of Caperquin where he spent a whole year in melancholy reflections on the vicissitudes of fortune. In the year 1649, he entered the army as a lieutenant, serving against the Catholics who were stirring up trouble in Munster. In 1656 he left the army and ever after lived upon his estate

[1] Read before the Johns Hopkins Hospital Historical Club, April 11, 1904.

at Affane. He was possessed of property, and was evidently held in some esteem for he became a justice of the peace and registrar for plantations.

How this quiet, contemplative, well-to-do country gentleman ever became possessed with the idea that he was gifted with the ability to heal disease by the touch of his hand will never be known. In 1662, however, he claimed to have discovered his supernatural gift, although for some time it appears he kept the virtue to himself, but at last, like the good man that he was, told his wife, who unkindly laughed at him. Nothing daunted, however, he began laying his hands upon various people in the neighborhood who suffered from scrofula or other diseases, and is said to have cured very many of them. These doings made such a noise, however, that they came to the ears of the authorities, and the Bishop's Court at Lismore prohibited him from laying on hands in the future. He seems to have disobeyed the court's injunctions. As is the case with so many quacks, he soon found titled victims. In January 1665-6 the Earl of Orrery brought him over to England in order that he might lay his hands upon the wife of Lord Conway, who suffered from chronic headaches. Although he was unsuccessful with this patient, he, nevertheless, seems to have performed cures in a number of persons that lived in her neighborhood, so much so that an account of his cures in Warwickshire was published by a Mr. Stubbe in which he stated "that Mr. Greatrakes was possessed of some peculiar temperament, as his body was composed of some particular ferments, the effluvia whereof being introduced, sometimes by a light, sometimes by a violent friction, restore the temperament of the debilitated parts, regenerate the blood, and dissipate the heterogeneous ferments out of the bodies of the diseases, by the eyes, nose, mouth, hands, and feet." Stubbe's account was written in the form of a letter to the famous Robert Boyle, Esq. The latter was extremely put out at being made party to what he was at first disposed to regard as the performances of a quack, but *mirabile dictu,* after witnessing some eight laying on of hands, even the great Boyle succumbed and publicly announced that he could vouch for some of the wonderful results achieved by the healer.

This English visit marked the climax of Greatrake's career. He made large sums of money, but within a few years his fame declined as rapidly as it had risen, and he died in obscurity.

Queen Anne's poor eyesight rendered her a godsend to quack ophthalmologists. Two of these rascals the Queen especially delighted to honor. One was a tailor named William Reade whom she knighted. The other was a shoemaker, named Roger Grant. He was made oculist to the queen by special appointment. The champion quack ophthalmologist of the eighteenth century, however, was undoubtedly the Chevalier John Taylor. Dr. Brown Pusey, of Chicago,[2] has recently published an account of his career founded upon his biography which was published in London in 1761, with the following title:

"The Life and Extraordinary History of the Chevalier, John Taylor, Ophthalmiator, Pontifical, Imperial and Royal; in two volumes; written from authentic materials and published by his son, John Taylor, Oculist; London, 1761." Dr. Pusey truly says, "in comparison with him the quacks of the present day are a cheap lot." Dr. Pusey gives extracts from the hand bills which it was his wont to distribute, and their glowing eloquence puts to shame all modern imitators. In the course of his book, he narrated the tremendous distances he had travelled, and the vast experience he had had. He was appointed Ophthalmiator Pontifical by Pope Benedict XIV, and was appointed oculist to George II, King of England, Augustus III, King of Poland, Frederick V, King of Denmark and Norway, Frederick Adolphus, King of Sweden. As Dr. Pusey says one of his important cases was in the person of a man who seems nearer to us than all these high personages, namely Handel, of whom he says, "I once thought to have had the same success, having all circumstances in his favor, motions of the pupil, light, etc., but on drawing the curtain, we found the bottom defective."

His son followed in his footsteps as a quack oculist, and although he did not achieve as wide a reputation as the father, he was probably more successful from a pecuniary point of view, achieving a large practice which he kept until the end of his days.

Goddard's Drops.

There have been quacks whose fame was based upon some nostrum, the recipe for which has since been revealed to the world, and in some instances the preparation is still in use, generally with modifications, or under a different name. Thus we seldom realize nowadays when we prescribe Aromatic Spirits of Ammonia that they were originally famous as a secret preparation known by the name of "Goddard's Drops." They were originated by Dr. Jonathan Goddard, a fellow of the Royal Society, and Professor of Medicine in Gresham College. Dr. Goddard had a large practice, and was a professional adviser as well as an intimate friend of Oliver Cromwell. He had a laboratory in Gresham College in which he manufactured various medicinal preparations, of which the drops became the most famous. He for some time kept their manufacture a secret, and sold them through apothecaries but as this was contrary to his oath to the College of Physicians, he was obliged to reveal his secret to them, but only did so upon their promise that the recipe should not be published until after his death. They were at first known as Spiritus Salis Volatalis Oleosus, or Sal Volatile Drops. In "Doctors and Doctors" by Graham Everitt, there is a quotation from the Sloane MSS. 958, in which there is an entry, in a memorandum book, in the handwriting of John Coniers, an apothecary on Shoe Lane which reads as follows:

"March 24, 1674-5. About ten o'clock that night my very good friend, Dr. Jonathan Goddard, reader of the physic lectures at Gresham College, suddenly fell down dead in the street, as he was entering a coach. He was a pretty corpulent and tall man, a bachelor between 45 and 50 years of age; he

[2] Journal of the American Medical Association, November 7, 1903.

was melancholy, inclined to be cynical, and used now and then to complain of giddiness in his head. He was an excellent mathematician, and some time physician to Oliver the Protector."

Spot Ward.

Another substance which is now in general use, but of which the nature was at first kept secret by its inventor, is oil of vitriol, or oil of sulphur. Joshua Ward, its discoverer, derived his nickname "Spot" Ward from a facial blemish. He was originally a chemist, and in the course of his work discovered a method of making oil of sulphur by the combustion of sulphur with saltpeter, the resulting compound being so cheap in its manufacture, that it soon completely supplanted the real oil of vitriol in the market. Ward was a thorough going quack, and employed every precaution to prevent the discovery of the nature of the substance which he used. He was a large, fat man, and used to drive around in a magnificent chariot drawn by four horses. He was high in the favor of George II, who provided him with a sort of dispensary and office in White Hall at which he attended the poor and gave them his medicines for nothing at the expense of the king.

Upon one occasion, when he had been paying a professional visit to his majesty, he turned his back on the king in leaving the room and started for the door. A court officer stepped forward and reminded him in a whisper that he should not turn his back on the king, to which he replied in a loud voice "The king's seeing of my back is a matter of no consequence, but the breaking of my neck falling backwards is of consequence not only to him but to the poor."[1]

Lady Mary W. Montague refers to Ward's preparation in a letter of 1748, and adds to it one of her usual keen reflections. "I find," she says, "that tar-water has succeeded to Ward's drops; and it is possible that some other form of quackery has by this time taken place of that. The English are, more than any other nation, infatuated by the prospect of universal medicine."

John St. John Long.

John St. John Long deserves to be ranked among the most successful quacks of all time. From his practice he is said to have derived an income of over £13,000 a year, and fortunately for him, death terminated his career at a time when many victims cherished a belief that he was the greatest of healers. He was the son of a poor Irish peasant, and passed his youth in the village of Doneraile. He was possessed of a natural talent for drawing and painting, and after passing a short time in Dublin, in the cultivation of these gifts, he established himself as an artist in Limerick. Here he painted portraits and gave instructions in painting and drawing to such pupils as he could procure. Finding the village of Limerick somewhat limited as a sphere for his activity, Long migrated to London where he worked for some time in the studio of Sir Thomas Lawrence. During this time he turned a few honest shillings by making anatomical drawings and paintings for the professors in the London Medical Schools. In the course of his work, he picked up some crumbs of medical knowledge, and soon announced that he had discovered a wonderful liniment by means of which he was not only able to cure most diseases, but also to discover the existence of latent disease in various organs. If, for example, this substance were applied over the chest of any person if they were healthy, and if there was no phthisical or other affection of the lungs, the application would be absolutely void of result. If, however, the lungs were in any way diseased, the substance would produce a counter irritation resulting in the formation of a weeping sore. The acrid substance which flowed from this, Long said was the essence of the disease. His enemies claimed that he used two distinct fluids to effect this wonderful result, one a bland and innoxious substance, the other a blistering liquid which originally bore the name of St. John Long's liniment.

In a footnote in the United States Pharmacopeia, the following instructions are given for making the latter:

"Take the yolk of one egg, 5 fluidrachms of acetic acid, 3 fluidounces of oil of turpentine, 2½ fluidrachms of rose water, and ½ fluidrachm of oil of lemon. Make an emulsion."

He also had a remedy in the form of vapor which was to be inhaled. For this purpose his patients would seat themselves around a large cabinet from which radiated a number of stems, through which they inhaled the vapor. Long fitted up magnificent offices in a mansion in Harley Street, and here the aristocracy flocked in swarms to inhale and to be rubbed. The list of Long's patients reads like a directory to the fashionable quarter of London. But not only was he patronized by the citizens of the metropolis, but the sick and the foolish flocked from every quarter of the United Kingdom. The contemporary accounts of some of the scenes in Long's offices, whether written by his friends or his enemies, are most amusing. It was only possible for him to see patients for a few moments, and he would then turn them over to one of his assistants, or else send them into the inhalation room to join the other patients sucking the miraculous vapor through Long's tubes.

Long seems to have had a most attractive personality. He was tall and well built, with a handsome face and agreeable manners. He lost no opportunity to ingratiate himself with his fashionable clientêle, and was soon a very welcome guest in some of the most fashionable houses in London. Particularly was he a favorite with the ladies, and more than one noble female is said to have offered him an opportunity to improve his fortunes by contracting a matrimonial alliance with her. It deserves to be recorded to his credit that no hint of scandalous relations was ever breathed against him, although his enemies would only have been too glad to have availed themselves of an opportunity to cast aspersions upon his character. Upon Long's death it was found that he left a widow in Ireland which accounted for the firmness with which he rejected the advances of other ladies.

[1] Professional Anecdotes or Ana of Medical Literature, London, 1825.

One way in which he enormously increased his practice was to persuade healthy persons that he saw indications of latent disease within their systems, which could be demonstrated by an application of his wonderful liniment. As most of his patients were accompanied by various female relatives and friends on their visits to his office it was very easy for Long to cast his net about the healthy while bestowing his professional attentions upon the sick. As we shall see, it was this trick of Long's which got him into his first serious encounter with the law when he practiced it upon the sister of a young Irish girl whom he was attending for phthisis.

In 1829, Long wrote a book which he published with the following title:

"Discoveries in the Science and Art of Healing. By John St. John Long, Esq., M. R. S. L., together with the evidence upon which the author claims the confidence of the country, etc., etc."

The front page was adorned with quotations from Lord Bacon and Sir Astley Cooper. The book was dedicated to Sir Richard Paul Jodrell, Bart., and in the course of a lengthy epistle to that nobleman, the wily author introduced a letter from Lord Ingestre. In the introduction he stated that the object of his work was "to lay before the Public the successful results of my researches on some of the more important diseases of the human frame, particularly in the cure of consumption and mania, and prevention of the fatal consequences of small-pox, measles, whooping-cough, or other analogous inflammable disorders to which I shall subjoin my reasons for departing from the established rules of medical practice." He claimed that he could show that all these various deviations from health were the result of presence of a "certain acrid matter or fluid pervading the system while in a state of disease." The book was also designed "to submit the documentary evidence upon which I claim the confidence of the country." Long affirms that no one can deny that in cases of illness, medicines of a poisonous and deleterious quality are frequently administered. He very judiciously quotes in a footnote a statement of Hunter, "We find that new diseases arise from mercury alone. The tonsils shall swell where no disease has been before. The periosteum shall thicken and also probably the bones, and the parts over these shall become œdematous and sore to the touch." Long's method of applying quotations is very similar to that of the man who pointed to the fact that the bible contained the assertion "There is no God," omitting the reference to the preceding verse, "The fool hath said in his heart," Mr. Long then wisely asks "what healthful union can there be between mercury, prussic acid, henbane, digitalis, acetate of lead, sulphuric acid, nitrous acid, and flesh and blood?" Having thus challenged the medical profession, Long proceeds to a series of observations on the origin of consumption and other diseases. This section consists in an exposition of his views concerning the "acrid matter" which is most amusing. He nowhere makes any attempt to explain the nature of this acrid matter, but confines himself to the dogmatic statement that practically every disease is the result of its presence. Thus he states "This acrid matter is inherent in the human frame, and is first apparent under the form of small-pox and measles, etc. The susceptibility to these diseases originates with men's existence, and according to circumstances is operated on, and brought into action at different ages. But although many persons may altogether escape exhibiting the usual external symptoms, yet the peccant substance is more or less in the system. In those cases, a portion of the acrid matter must have exuded from the system, still leaving a sufficient quantity to operate in future time, and under a new form."

In regard to cholera, diarrhœa and dysenteric affections he states, "My power over this class of diseases is so great, that I am desirous of communicating the knowledge to the Honourable the East India Company. I shall be happy to show them the control I possess over every species of intestinal irritation; and over maladies that may in their worst states and stages immediately be cured. So simple is the process, that every individual in India may always carry the remedies about his person, so as to use them at the moment of the attack." In the same manner such varied infections as liver complaints, gout, cataract, deafness, erysipelas, ring-worm are all of them claimed to yield readily upon Dr. Long's wonderful remedy extracting the acrid matter from the constitution.

In the treatment of insanity to which the next section of the book is devoted, Long claims to have been particularly successful. The following chapter is devoted to an outline of his general method of treatment. He states that his general practice consists of applications and certain fumigations so innoxious and gentle in their nature, that they may be employed either by himself or by the patients without producing any unpleasant effects whatsoever. In this section there are one or two judicious quotations from Hippocrates.

The concluding section of the work is composed of letters from grateful victims. They possess a wonderful resemblance to that chapter of the "Key to Health and Science of the Scriptures" which Mrs. Eddy has entitled "Fruitage," which an irreverent reader has suggested should be read "Easy Fruitage."

On Saturday, October 30, 1830, Long was brought to trial at Old Bailey charged with manslaughter in occasioning the death of Catherine Cashin. He pleaded not guilty to the indictment. The facts elicited at his trial were as follows:

Miss Catherine Cashin was a young lady of good family in Ireland who had brought her younger sister up to London to be treated for phthisis. The elder sister, at the time of her arrival in London, was in perfect health. She had taken her sister a few times to Long's house, when the quack announced that he had discovered signs in her of latent consumption, and that if she did not put herself promptly under his care, she would develop the disease within two months. This frightened the young woman to such an extent that she agreed to place herself in his hands. Accordingly, on the 3rd of August, she went to his offices and was rubbed with the famous liniment between her shoulders and on her back. Within a short time the parts began to ulcerate and become

gangrenous and she died on the 17th of the month. Before her death Mr., afterwards Sir Benjamin Brodie was called in and at the trial he gave it as his opinion that her death was the result of the mortified sore upon her back. At the autopsy there was no evidence of disease of any of the organs, save the large sloughing sore between the woman's shoulders. The woman employee of Long, who performed the actual rubbing on Miss Cashin's back, testified that she had rubbed the woman with the same lotion that had been used on a number of other persons, among them the Marchioness of Ormonde and Lady Harriet Butler. The wound was dressed with a cabbage leaf, and treated in a precisely similar manner to that of the other patients. A number of witnesses were called in in behalf of the prisoner, amongst them some of the best known people and of the very highest social standing in London. The judges charge was strongly in favor of the prisoner, though the jury found him guilty. His punishment, however, was not excessive, as he was only fined £250, which sum he pulled from his pocket in the court room. The account of the trial concludes, "having quitted the bar, he proceeded to the court yard in company with his friends, where they got into a curricle of the Marquis of Sligo and rode off with his Lordship amidst the clapping of his noble friends, and the hootings and hissing and laughter of the populace."

On November 10, 1830, Long was again brought before a coroner's jury, accused of manslaughter in having caused the death of Mrs. Colin Campbell Lloyd, the wife of a captain in the navy. Her medical attendant and surgeon named George Vance, stated that he had been called in to see Mrs. Lloyd, and found her suffering from a large sloughing sore, covering practically the entire front of her thorax, and extending down so far that the sternum was found bare. She stated that she had been rubbed twice at St. John Long's house with his liniment. Mr. Brodie who saw her stated that the appearance of the sore was exactly similar to that which he had found on the back of Miss Cashin. Another surgeon, a Mr. Campbell, testified that he had seen Mrs. Lloyd some days before she had consulted Dr. Long, and that she had been in perfect health. She had visited the quack with a view to procuring relief from a slight throat infection. After examining her, Long had informed her that the throat trouble arose from extensive disease of the lungs, they being full of small ulcers, and recommended his inhalation treatment in addition to applications of his liniment to her chest. Her husband likewise testified that she had been in perfectly good health until after the rubbings at Long's. At the autopsy, all Mrs. Lloyd's organs were perfectly normal, and the body showed no signs of disease, with the exception of the enormous sloughing sores over the chest and upper part of the abdomen. The jury found that the deceased had come to her death as the result of the application of a liniment to her chest by Dr. Long. When brought to the trial at the Old Bailey on the charge of having killed Mrs. Lloyd, the jury acquitted him.

Long's quackery was, of course, grist to the mill of Thomas Wakely, the editor of "The Lancet," and his editorials during the years 1830-31 upon the subject of St. John Long, and the legal procedings against him are most interesting reading. Amongst those who testified to the efficacy and safety of St. John Long's practice was Sir Francis Burdett, Bart., Wakely gives the following quaint narrative of the occasion of Burdett's resort to the quack. He writes: "The motive which induced Sir Francis Burdett to first visit Long has been thus explained. The honorable baronet is on terms of intimacy with the Marquis of Anglesea, who left the plains of Waterloo minus a leg. Sir Francis, keenly feeling for his friend's misfortune, having heard of the miraculous powers of the mystic "liquid" and having read probably that if the claw of a lobster be taken off, the energies of the animal are equal to the production of another claw, he applied, it is said to Long to know whether, if the "secret fluid" were gently "rubbed" over the skin of the stump, the marquis' leg and foot might not grow again? It is reported that although the leg and foot were not forthcoming, the operation was not entirely unproductive, having within a short period produced a great toe."

Among St. John Long's most ardent defenders was Dr. Ramadge, of Ely Place, London. He wrote a long letter addressed to Long, advocating his cause which was published in the London Sunday Times, on April 10, 1831, in which he stated that he had been at first an opponent of Long's but that after deep reflection he had changed his opinion. He claims that at the autopsy on Miss Cashin there were found distinct evidences of pulmonary disease, and that the severe effects produced by the liniment "occurred because she had overloaded her stomach with purple grapes and plums, causing febrile attacks, and thereby aggravating the sore on her back."

Wakely republishes Dr. Ramadge's letter in "The Lancet" of April 16, 1831, accompanying it by the most severe editorial comments on his unprofessional behavior, from which we would only quote the following:

"If, in short, it be possible for Dr. Ramadge to be the author of this letter, we advise him, forthwith, to abandon the ranks of a profession which he has disgraced, or most assuredly the members of the profession will abandon him."

In spite of all the expositions of Long's quackery which were published not only in the medical but in the lay press, he continued to be upheld by a large and influential clientêle. His career was terminated at the early age of 37 by phthisis pulmonalis, the very disease to which he had laid his most extravagant claims to cure. At his death it was found that as a result of his nefarious practice, he had accumulated a snug fortune which he left to his widow in Ireland. He was buried in Kensal Green Cemetery, and over his grave some of his admiring victims erected an elegant monument which bore the following unique inscription:

"It is the fate of most men
To have many enemies, and few friends.
This monumental pile
Is not intended to mark the career,
But to shew
How much its inhabitant was respected
By those who knew his worth,
And the benefits
Derived from his remedial discovery.
He is now at rest,
And far beyond the praises or censures
Of this world.
Stranger, as you respect the receptacle of the dead
(As one of the many who will rest here),
Read the name of
John Saint John Long
without comment."

DR. PERKINS.

Among successful American quacks the name of Elisha Perkins should always hold a preëminent position as the inventor of the famous Tractors which convulsed the medical profession not only in this country, but throughout the continent of Europe. He came of an eminently respectable New England family. His father, Joseph Perkins, was a native of Norwich, Conn., and his mother was a daughter of another physician of the same town, Dr. Caleb Bushnell. Dr. Joseph Perkins had graduated from Yale in 1727, being the first of thirty-six of that name who took their degrees at Yale College between 1727 and 1858. After graduating he studied medicine, and settled down to practice in his native town. He is said to have performed more capital operations than any other medical man in that part of the colony in Connecticut. He is frequently spoken of in contemporary articles as a man of brilliancy; he was also possessed of a pious disposition, a deacon in the church, and an enthusiastic worker in benevolent enterprises. He died in 1794, in the ninetieth year of his age. It is painful to think what must have been his reflections, if he were indeed a man of brilliancy and piety, upon the successful quackery of his son.

Elisha Perkins was born at Norwich, Conn., on January 16, 1741. He not only received his preliminary education from his father, but also studied at Yale. He received paternal instruction in medicine, and began his medical career by serving as his father's assistant. After devoting some years to medical practice in this capacity, he settled down to practice at Plainfield, Conn., where he rapidly acquired an extensive business. It is said that he frequently rode sixty miles a day on horseback in the course of his professional visits. He was well fitted for the work, being a man of remarkably vigorous constitution and great physical strength. He was above six feet in height, and excellently proportioned; he was accustomed to sleep but three or four hours in the twenty-four, and was a total abstainer. According to contemporary accounts, this paragon was of the most kind and sympathetic disposition. It is an unfortunate thing that most of our information concerning him is based upon eulogistic obituary notices, or derived from contemporary endorsements of his victims, two forms of biography which must give special pleasure to the Father of Lies. It is curious to find an account of Perkins inserted in the eminently respectable Thatcher's American Medical biography, and yet more strange to find that it consists chiefly of information derived from the glowing notices above mentioned, and dealing very gently with the element of quackery which entered so largely into its subject's practice.

In his son's pamphlet describing the Tractors, the following account is given of the way in which Dr. Perkins was first led to the discovery that there was an occult therapeutic property innate in certain metallic substances. "The first remarkable incident that presented itself to the notice of Dr. Perkins, was the sudden contraction of a muscle, when he was performing a chirurgical operation. This he observed regularly took place whenever the point of the metallic instrument was put in contact with the muscle. Struck with the novelty of the appearance, he was induced to try the points of wood, and other substances; and no contractions taking place on these experiments, he thence inferred that the phenomena could be ascribable only to the influence of the metal.

"About the same time he observed, that in one or two cases, a cessation of pain had ensued when a knife or lancet was applied to separate the gum from a tooth, preparatory to extracting it; and in the same year he discovered, that momentary ease was given in a few instances, by the accidental application of a metallic instrument to inflamed and painful tumours, previous to any incision." He then proceeds to describe how Dr. Elisha Perkins for some years experimented extensively with instruments made of various metals with the object of ascertaining more of their hidden properties, and as the son writes "The result corroborated and indeed exceeded his most sanguine expectations; for he discovered that, by drawing over the parts affected in particular directions certain instruments which he formed from metallic substances into certain shapes, he could remove rheumatism, gouty affections, pleurisies, inflammations in the eyes, erysipelas, and tetters; violent spasmodic convulsions, as epileptic fits; the locked jaw; the pain and swelling attending contusions; inflammatory tumours; the violent pains occasioned by a recent sprain; the painful effects of a burn or scald; pains in the head, teeth, ears, breast, side, back and limbs; and indeed most kinds of painful topical affections, which came under his care and observation. The instruments producing these effects are termed TRACTORS."

The instruments as finally made by Dr. Perkins consisted of two pieces of metal, about three inches in length, each pointed at one end, and in appearance greatly resembling horse shoe nails. They were utilized by drawing them gently over the part which it is desired to affect, the stroking generally lasting for some ten or twenty minutes at a time. A great deal of importance was attached to the treatment being carried out according to the printed instructions which were furnished with each pair, but it was most important of all that nothing but Dr. Perkins' Tractors should be used; no other pieces of metal would suffice, save those which were marked

with the trade mark which guaranteed them genuine. They were retailed at five guineas a pair.

There is an amusing print by Gilray, representing a Perkinean doctor making an application of the Tractors to the pulpy red nose of an alcoholic Britisher. The victim seems to be in a most uncomfortable frame of mind; he is in an attitude expressive of the highest nervous tension. The doctor is steadying his head by holding his bald pate with one hand, having pushed his wig over his arm chair. John Bull's teeth and fists are clenched, and he is evidently in a terribly nervous state of apprehension, nevertheless, he has no intention of giving up his bad habits, for on the table beside him are brandy, sugar, lemon and hot water, all ready to be incorporated into a reeking punch. On the wall of the room we can just discern the fat legs of a little Bacchus, sitting astride a barrel. The apostle of Perkins is most intent upon his operation. He is applying but one of the Tractors, the other he holds between his teeth. We fear that Gilray would not have been a successful practitioner, as all the directions for their use distinctly state their valuable influence is exerted through the transmission of animal electricity from one tractor or pole to the other through the patient's tissues. It is probable, however, that the caricaturist purposely intended to misrepresent the method of application in this instance.

It is gratifying to learn that this delusion made less headway in America than it did when exploited in London and in other cities on the Continent. Elisha Perkins sent his son, Benjamin Douglas Perkins, abroad for the purpose of diffusing his propaganda in other lands. The success which he achieved is almost incredible. An institution which rejoiced in the name of The Perkinean Institution was founded in London under the presidency of Lord Rivers, the object of which was to furnish free treatment to the poor. Upon the list of those who endorsed the institution we find no less than eight professors of various branches of medicine and surgery, forty physicians and surgeons, and thirty clergymen. In Copenhagen the success of the Tractors was fully as great. The physicians on the staff of the Royal Frederick Hospital in that city published a long series of experiments with the Tractors, accompanied by reports of miraculous cures which had been wrought by their means.

In 1799 Benjamin Douglas Perkins, who was then residing in London, published a book entitled:

"Experiments with the Metallic Tractors, in Rheumatic and Gouty Affections, Inflammations, and various Topical Diseases, published by Surgeons Herholdt and Rafn, of the Royal Academy of Sciences, Copenhagen; Translated into German by Professor Tode, Physician to his Danish Majesty, Thence to the English Language by Mr. Charles Kampfmuller; also Reports of about one hundred and fifty cases, in England, demonstrating the efficacy of the Metallic Practice, In a Variety of Complaints, both upon the Human Body, and on Horses, etc., By Medical and other Respectable characters. Edited by Benjamin Douglas Perkins, A. M., of Leicester-Square, London, Son of the Discoverer."

The last paragraph of the introduction is most ingeniously suggestive, it reads:

"The Tractors with printed Directions for their use in families, may be had of the Editor, at his house in Leicester Square, price Five Guineas the set.—To prevent frauds and impositions, every genuine set of the Tractors is stamped with the words 'PERKINS PATENT TRACTORS;' and to the paper of directions, attending them, is subjoined a receipt for the Five Guineas, numbered and signed in his handwriting; thus 'Benjamin Douglas Perkins.' The hours of attendance on Patients at home are from Nine to Three o'clock in the Morning, The remainder of the day is devoted to Patients abroad."

Benjamin Douglas Perkins in the same year also published, while still in London a pamphlet entitled:

"The Influence of Metallic Tractors on the Human Body in Removing various painful Inflammatory Diseases, such as Rheumatism, Pleurisy, Some Gouty Affections, etc., etc., lately discovered by Dr. Perkins, of North America; and demonstrated in a series of Experiments and Observations, by Professors Meigs, Woodward, Rogers, etc., etc., by which the importance of the Discovery is fully ascertained, and a new field of Enquiry opened in the Modern Sciences of Galvanism, or Animal Electricity. By Benjamin Douglas Perkins, A. M. Son of the Discoverer."

The excitement which prevailed over this wonderful discovery lasted well into the first decade of the nineteenth century, but it then gradually subsided, and in a few years the Perkins' golden harvest was over. Thatcher, with characteristic conservatism concludes his account of Perkinism with the following paragraph:

"Such is the history of the Metallic Tractors. It is to be considered a singular and unaccountable circumstance that the remedy should have been consigned to oblivion. Is it within the bounds of probability that the vast amount of authenticated evidence that has been produced, should be resolved into a delusion, a mere phantom of the imagination? However enthusiastic the promoters of the novel discovery may have been, it is more clear that autoptical evidence was in every instance at command, and might have precluded the possibility of deception. If, therefore, Perkinism has shared the fate of Animal Magnetism, there is no sufficient reason for supposing that its discoverers should be placed in the same class with Mesmer, as he undoubtedly was a man of honorable principles and character."

It is interesting to read that while foreign nations were adding their tribute of homage to Perkinism, Elisha Perkins was expelled in 1797 from the Windham County Medical Society of which he had been a member, and in 1795 the chairman. In 1799 there was an epidemic of yellow fever in the city of New York. Elisha Perkins had some time before brought forward what he claimed was a specific remedy in the treatment of diphtheria, typhoid fever, and dysentery. The mixture consisted of sodium chloride dissolved in vinegar. This was diluted with hot water. Anxious to prove its efficacy, he hastened to the smitten city where he fell a victim to the disease, and died a few weeks after his arrival in the fifty-ninth year of his age. It was subsequently said of him, as of a more famous character in history, that nothing in his life became him so well as the leaving of it. We believe this to

be an erroneous view, Perkins was a quack, pure and simple, and only went to New York with the idea of exploiting himself yet further in the public eye. As to his son, Benjamin Douglas, after graduating from Yale in 1797, he had gone to London to preach the doctrine of the Tractors. He accumulated an enormous fortune with which he returned to New York and opened up a bookstore in partnership with a man named Collins. In this enterprise, he proved very successful. Towards the latter part of his life, his religiosity became very prominent, and he became a strict quaker. Thatcher says: "He sustained a character of inestimable worth; was frank, honorable and discrete; in morals exemplary; in religion undissembling and devout."

Among the letters contained in the book which Benjamin Douglas Perkins edited, was one from John Vaughan, M. D., M. P. M. S., in which he narrated some wonderful cures effected by the use of the Tractors and the book also contains a letter from John Tilton, President of the Medical Society in the State of Delaware, which contains the following remarkable statement:

"I am glad you have at length overcome the restraints of diffidence, by giving us your thoughts on Electricity, in explanation of Dr. Perkins' Metallic Operation. That some general principle exists, which gives metals a powerful influence on the animal economy, is now universally acknowledged by philosophers the most candid and best qualified to judge. Instead of involving Dr. Perkins in the disgrace of Mesmer, I apprehend we ought rather to contrast the honest man with the rogue; for, without art or affectation, Dr. Perkins barely discloses useful facts, by means the most simple and easy.

It must be confessed, however, that many reject our general principle: like infidels to the gospel, they admit of no mysteries, and refuse to believe what they do not readily comprehend. Others who acknowledge that a general principle pervades the animal economy, on which the metals have influence, nevertheless, dispute about what this notable power may be. Both these classes of men ought to thank you. To the one you have given a reason they did not apprehend, and the other should wish for the best account known or well understood. For my part, I frankly confess, I shall be satisfied with the principle of ANIMAL ELECTRICITY, until the Phenomena are better explained by means of some other."

Tilton was one of the foremost physicians of his time. He was a graduate of the first medical class in the University of Pennsylvania, and had been offered the chair of Materia Medica in that institution. He had had an honorable career as an army surgeon during the Revolutionary war, and wrote a very valuable little book entitled "Observations on Military Hospitals," and in the war of 1812, he was physician and surgeon-general in the United States Army. It is not much wonder that the Perkineans were delighted to obtain anything like an endorsement from such a man, and it is no wonder that if Dr. Tilton was inclined to think there was some value in the metallic Tractors, other persons should be deceived.

ON THE CARDIAC AND VASCULAR COMPLICATIONS AND SEQUELS OF TYPHOID FEVER.

THE JEROME COCHRAN LECTURE.[1]

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[1] Delivered before the Alabama State Medical Association at Mobile on April 20, 1904, and published in the Mobile Medical and Surgical Journal for July, 1904.

I. INTRODUCTORY REMARKS.

On receiving your very kind invitation to speak before you to-day, it seemed to me that the most profitable subject to discuss would be that in the study of which I was at the moment engaged, the cardiac and vascular complications and sequels of typhoid fever.

A glance at the extensive literature concerning this question cannot fail to impress one with the considerable variance of opinion existing between different observers as to the frequency and extent to which the heart and vessels suffer in this malady.

(1) THE HEART.

(a) *Endocarditis.*—Concerning the frequency of endocarditis as a complication of typhoid fever there is little difference of opinion. Most observers agree in regarding it as a rare and unimportant complication. Among the 2000 Munich necropsies[2] there were but 11 instances of endocarditis. Girode,[3]

[2] Hölscher: München. med. Wchnschr., 1891, XXXVIII, 43, 62.
[3] Bull. méd., 1889, III, 1392.

Viti,[4] Carbone,[5] and Vincent,[6] have obtained typhoid bacilli from endocarditic vegetations and Lion,[7] has produced experimentally, an endocarditis of the aortic valves in rabbits after intra-venous injection of typhoid cultures. The majority, however, of the cases of endocarditis which occur as complications of typhoid fever are probably the result of secondary infection.

(b) *Pericarditis.*—The same is true—to an even greater extent—of pericarditis.

(c) *Myocarditis.*—With regard to the myocardial changes, there is a most extensive literature dating from the early part of the last century. The recent investigations of Romberg,[8] Landouzy and Siredey,[9] Renaut,[10] Mollard and Régaud,[11 12] Giacomelli[13] and others have shown clearly that in some instances the changes may be sufficient to play a grave part in determining the fatal result of the disease. Moreover, the extent of these lesions is sometimes such that it can scarcely be doubted that in cases which recover, grave sequels must appear in after years. Great differences of opinion, however, exist both as to the relative frequency of acute myocarditis, its relation to the clinical manifestations of the disease and the frequency with which it results in serious permanent damage to the heart.

What clinical symptoms justify us in assuming the existence of an acute myocarditis?

How often does typhoid fever result in permanent damage to the heart?

These are questions with regard to which there is the widest difference of opinion.

It must be acknowledged that this disease has not in the past been regarded as an important agent in the production of chronic cardiac or vascular defects. A few observers, however, have been outspoken in their opinion that typhoid fever has an influence of real significance in the production of acute and chronic myocarditis. Chief among these have been Landouzy and Siredey and Mollard and Régaud.

The emphasis which the first of these observers places upon the effect of typhoid fever upon the heart and vessels is strikingly shown by the following words from a communication made some eighteen years ago.[14] "The day when hygiene shall prevent typhoid fever, it will not only save from death each year more than one thousand Parisians, it will save more than a thousand individuals from disease of various systems, especially from diseases of the heart, that is, it will save each year at least a thousand invalids."

These are striking words. Are they justified by fact? There are in literature few, if any, systematic clinical observations with regard to the point.

(2) The Arteries.

A survey of the literature with regard to affections of the arteries in typhoid fever is of considerable interest. Little attention has been paid in this country to the occurrence or significance of acute arteritis in typhoid fever, to its influence upon the course of the disease or to its later sequels. The chapter on typhoid arteritis and arterial thrombosis in most text-books on typhoid fever is extremely short; yet there is a large and interesting literature upon the subject.[15] Gangrene of the extremities has long been recognized as an occasional complication of the disease and the fact that this is in many instances due to autochthonous thrombosis associated with an acute arteritis has also been pointed out.

That acute endarteritis may occur in the aorta and smaller arteries in connection with typhoid fever as well as in other acute infections is mentioned by many observers, and yet its extent, its frequency, its possible influence in determining grave local or general disturbances in later years, have not to my knowledge been systematically investigated.

Curschmann[16] in his recent work says: "I would emphasize expressly that in a large number of post-mortem examinations in cases of typhoid fever in which I have noted the condition of the coronary arteries I have never found these diseased"—a statement considerably at variance with our experience.

(3) The Veins.

With regard to the venous complications of typhoid fever much more has been written and much more is known. The venous thromboses so frequent especially in the lower extremities, which since the time of Virchow, have been regarded as essentially marantic, are now again more generally considered as secondary to an actual phlebitis due in some instances to the typhoid bacilli alone, in others to secondary, mixed

[4] Atti d. r. Accad. d. fisiocrit. di Siena, 1890, 4 S., II, 109.

[5] Gaz. med. di Torino, 1891, XLII, 122.

[6] Mercredi méd., 1892, III, 130.

[7] Essai sur la nature des endocardites infectieuses. 4°, Par., 1890.

[8] Uber die Erkrankungen des Herzmuskels bei Typhus abdominalis, Scharlach und Diphtherie. Deutsch. Arch. f. klin. Med., 1891, XLVIII, 369; 1892, XLIX, 413.

[9] Contribution à l'histoire de l'artérite typhoïdique; de ses conséquences hâtives (mort subite) et tardives (myocardite sclércuse) du cœur. Rev. de méd., Paris, 1885, V, 843. Landouzy: La fièvre typhoïde dans ses rapports avec l'appareil vasculaire et cardiaque. Gaz. d. hôp., Paris, 1886, LIX, 323. Landouzy et Siredey: Etude sur les localizations angiocardiaques typhoïdiques, leurs conséquences immediates, prochaines et éloignées. Rev. de méd., Paris, 1887, VII, 804, 919.

[10] Les myocardites aiguës. Congrès Français de médecine. V. session, Lille, 1899; Paris, 1899, II, 1-83.

[11] Etat des artères du cœur dans les myocardites aiguës. Congrès Français de méd., 1899, V, 280.

[12] Les troubles cardiaques dans la convalescence de la fièvre typhoïde. Presse méd., Paris, 1900, I, 19-22.

[13] Il miocardio nelle infezioni, intossicazioni, avvelenamenti. Ricerche anatomo-patologiche e sperimentali, Policlinico. Roma, 1901, VIII, M., 145-155.

[14] Landouzy: *Op. cit.*, 1886.

[15] For references see: Thayer. On Arteritis and Arterial Thrombosis in Typhoid Fever. The New York State Journal of Medicine, 1903, III, 21.

[16] Curschmann: Der Unterleibstyphus. 8°. Wien, 1898.

infections. Haushalter,[17] obtained typhoid bacilli in pure culture from the inflamed wall of the femoral vein, and was able to demonstrate them locally in the external coat of the vessel.

In such cases it has been generally assumed that the exciting cause of the thrombosis was afforded by the mechanical effect of actual anatomical lesions in the vessel wall. That other influences than purely mechanical changes in the wall may play an important part in inducing thrombosis in both arteritis and phlebitis has been shown by Flexner[18] and others and is borne out by my own observations.[19]

* * *

In view of these facts, it occurred to me that light upon some of the unsettled points might be obtained by a study of the material which has accumulated in the last fourteen and a half years in the clinic of Professor Osler at the Johns Hopkins Hospital and in the laboratory of the Pathological Department.

The plan which I laid out for myself was as follows:

(1) A study of the clinical records with regard especially to the cardiac and vascular symptoms and complications.

(2) A consideration of the anatomical records of those fatal cases in which necropsies were made.

(3) An investigation into the condition of as many of our old patients as could be examined.

At the outset I quite realized that the fulfilling of this whole task which should involve a careful histological study of our anatomical material, was more than could be accomplished in the time at my disposal. It has, however, seemed to me that an analysis of the question from a standpoint of clinical observation and a study of the gross pathological lesions could not fail to reveal facts of value and importance. These considerations I have the honor to present to you to-day.

II. General Clinical Analysis.

In the period between the fifteenth of May, 1899, and the first of January, 1904, there were admitted to the Johns Hopkins Hospital, 1458 cases of typhoid fever.

Of these 132 or 9.05 per cent died.

This rather high percentage is due to the fact that a considerable number of cases were brought to the hospital in a practically moribund condition.

In analyzing the clinical records of these cases, most of which I have had the good fortune to observe personally, I have selected for consideration those of the common cardio-vascular manifestations which might be regarded as *possibly* indicative of actual anatomical involvement of the heart or vessels, as well as the more definite vascular complications.

[17] Phlegmasia alba dolens et bacille typhique dans la fièvre typhoïde. Mercredi méd., 1893, IV, 126.

[18] Univ. of Pennsylvania Med. Bull., 1902, XV, 324.

[19] Thayer: On Arteritis and Arterial Thrombosis in Typhoid Fever. The New York State Journal of Medicine, 1903, III, 21.

(1) The Pulse.

(a) *Rapidity.*—Of these 1458 cases there were 220 or 15 per cent in which the pulse was above 140. 46 of these patients were under 15 years of age.

Of the 174 cases above fifteen years of age, 86 or nearly 50 per cent, resulted fatally.

77 cases showed a pulse of over 150; of these 55 or 71.4 per cent resulted fatally.

In 51 cases there was a registered pulse of above 160, with a mortality of 35 or 78 per cent.

In 6 cases there was a pulse above 170; two-thirds of these were fatal.

In 2 instances a pulse of 180 or above was recorded; one of these recovered.

In 2 instances the pulse was uncountable; both patients died.

(b) *Bradycardia.*—In 117 cases a pulse rate below 50 was noted. In all of these instances the bradycardia was a feature of convalescence. In one case only did a relapse follow.

In 6 cases a pulse below 40 was noted; the lowest recorded pulse rate—observed in two cases,—was 35.

All these 117 patients recovered. This circumstance when considered in connection with the fact that eight of these patients examined at periods varying from five months to eight years after their illness, showed no cardio-vascular defects, justifies the conclusion that the bradycardia of convalescence is of little or no prognostic significance.

(c) *Arhythmia.*—Irregularity,[20] of the pulse was noted in 29 instances, of which 10 resulted fatally.

In many of these cases the irregularity was associated with extremely rapid action of the heart and grave general symptoms.

Of 13 instances where the pulse was above 140, 9 or 69.2 per cent ended fatally.

In the remaining 16 cases, the irregularity was noted in several instances at the height of the disease; one of these resulted fatally. In the majority of cases, however, it was a feature of convalescence, being associated in 4 instances with a pulse of under 50.

(2) Heart Sounds.

Of the 1458 cases the heart sounds were clear throughout in 1125 patients. In 333 instances, or 22.8 per cent of the cases, cardiac murmurs were heard at one time or another.

In sixteen patients the murmurs were regarded as evidence of pre-existing valvular disease of the heart.

The lesions in these cases were as follows:

Mitral insufficiency	10
Mitral insufficiency and adherent pericardium	1
Mitral stenosis	2
Mitral stenosis and insufficiency	1
Aortic insufficiency	2
	16

[20] It is evident from the remarkably low figures that only instances of rather striking irregularity were recorded.

In addition to these cases there was one instance of Graves' disease with tachycardia and a systolic murmur at the apex.

Of the sixteen cases of valvular disease of the heart but 2 or 12.5 per cent died.

In the remaining 316 cases the murmur was systolic in 312 instances, diastolic once, while in 3 cases both systolic and diastolic murmurs were audible.

Analysis of the Cases in which Systolic Murmurs Alone were Heard.

Out of the 315 cases in which systolic murmurs were heard, there were two in which the record as to the localization of the murmurs was not satisfactory.

In the remaining 313 cases the murmur was heard:

At the apex alone in	87 cases.
At all four orifices in	66 "
At the apex, tricuspid and pulmonic areas in	49 "
At the pulmonic area alone in	35 "
At the aortic and pulmonic areas in	21 "
At the pulmonic area and apex in	20 "
At the apex, pulmonic and aortic areas in	11 "
At the apex and tricuspid areas in	9 "
At the pulmonic and tricuspid areas in	7 "
At the aortic, pulmonic and tricuspid areas in	4 "
At the aortic area alone in	1 "
At the apex and aortic areas in	1 "
At the tricuspid area alone in	1 "
At the apex, aortic and tricuspid areas in	1 "
	313 "

Cases in which the Systolic Murmur was Heard at the Apex.

There were 244 cases in which the systolic murmur was heard at the apex of the heart.

In 56 of these cases it was transmitted outward as far as the anterior axillary line.

In but one case was it noted that the murmur was audible in the back.

Time at which the Systolic Murmur at the Apex was First Observed.

In all of these cases the most detailed note was made usually upon admission to the hospital or within the several following days. Unfortunately, in many instances, careful notes upon the heart were not made later, and only in a rather small fraction of the cases was there a final record upon the day of discharge. It is, therefore, impossible to say with accuracy at just what period in the disease the murmurs developed. As a result of the fact that the first note was usually the most careful, the proportion of cases in which the murmur was audible on entrance is probably unduly high.

This was the case in 199 of the 244 cases.

The period of the disease at which the systolic murmur at the apex was first noted is illustrated by the following table:

Table I.

Illustrating the Period at which the Systolic Murmur at the Apex was First Observed.

	Weeks.										
Prodromal period	1st,	2nd,	3rd,	4th,	5th,	6th,	7th,	8th,	9th,	10th,	? Total
2	78	92	38	22	4	1	2	3	0	1	1 = 244

The table shows, as one might expect, that apical systolic murmurs are present with the greatest frequency during the height of the disease.

The following table shows the time of development of the murmur in 45 cases in which an apical systolic murmur, absent on admission, appeared later in the course of the disease.

Table II.

Showing the Time of Development of the Apical Systolic Murmur in 45 Cases in which, on Entrance, the Heart Sounds were Clear.

Weeks.									
1st,	2nd,	3rd,	4th,	5th,	6th,	7th,	8th,	9th,	10th
6	11	9	11	2	1	2	2	0	1 = 45

The figures in this table are closely similar to those preceding. The slightly greater prevalence of murmurs in the first weeks in Table I is doubtless due to the more careful initial examinations as observed above.

Of the 244 cases in which an apical systolic murmur was heard, this murmur disappeared before discharge in 107 instances; it was present upon discharge in 31[a] cases; there was no note in 106 cases.

Of the 45 cases in which the systolic murmur at the apex developed in the hospital this murmur disappeared on or before discharge in 17 cases; it was present on discharge in 10 cases; there was no final note in 13 cases.

Character of the Murmur in those Cases in which it Persisted upon Discharge from the Hospital.

In the 31 cases in which an apical systolic murmur was observed during the attack persisting in some form on discharge the murmur remained at the apex in 28 instances; in one instance it was audible only in the pulmonic area; in one, in the aortic, and in one, at both areas at the base.

In 2 of the 28 cases where the apical murmur persisted on discharge, the sound was louder at the base; in 1 instance it was louder over the right ventricle; once it was loudest in the aortic area—a clear case of aortic stenosis. Two further cases left rather early in the course of the disease on the day on which the note was made. In 3 cases the murmur on discharge was transmitted to the axilla.

In 10 cases a systolic murmur at the apex developed after admission to the hospital and persisted in the same area at the time of discharge; in one of these instances, however, it was more marked in the pulmonic area.

There can be little doubt that the cause of these apical systolic murmurs is to be sought, in the majority of cases, in the relaxation of the heart muscle with consequent dilatation

[a] In one of these instances the patient left on the day of entrance.

of the mitral ring. But the possibility that a certain proportion may be instances of endocarditis must not be lost sight of. The fact which will be mentioned shortly, that out of 132 necropsies there have been three instances of acute endocarditis, all unsuspected *intra vitam*, should be sufficient to convince one of the necessity of considering this possibility in every instance where cardiac murmurs persist after recovery from typhoid fever.

The recognition of this fact should suggest to the physician the wisdom of keeping old typhoid patients under observation during a considerable period following the disease just as in the case of maladies more commonly associated with endocarditis.

Basic Systolic Murmurs.

The cause of the basic systolic murmurs can hardly be discussed at length in a communication of this nature. Their frequency and slight prognostic significance are well known. One may, perhaps, recall the happy comment made, I think, by Broadbent, who after considering the anatomical and physiological relations of the aortic and pulmonic orifices, observed that it is not the fact that systolic murmurs are so frequently heard in these regions that is remarkable; it is rather that they are not always present.

In but one case (No. 44724), was the basic systolic murmur regarded as indicative of organic lesion. This was a clear example of aortic stenosis.

Diastolic Murmurs.

In 4 instances (Nos. 4020, 18118, 36484 and 41270), diastolic murmurs were audible.

Case I (No. 4020). The first case was that of a man, 20 years of age, without history of previous illness. On entrance a faint systolic whiff was heard at the pulmonic cartilage. The case was one of considerable severity. On the 27th day of his illness a soft diastolic murmur was heard in the third left space at the sternal border. Two days later the following note was made: "The diastolic murmur noted the other day is very audible in the second and third interspaces, not at the aortic cartilage. There is a soft systolic just within the apex." The patient made a slow but complete recovery; unfortunately no further notes were made on his heart. It has been impossible to trace the subsequent history.

Case II (No. 18118). The second case was that of a man of 19 who entered the hospital on the eighth day of a severe typhoid fever; on entrance his heart sounds were clear. On the twelfth day Dr. Osler noted that a soft bruit was heard along the left sternal border. On the 18th day, with a very rapid pulse, a soft diastolic murmur was heard at the aortic cartilage. Unfortunately, no further note was made upon the heart. The patient died four days later, of perforation. At necropsy no abnormalities were noted in the aortic valves. The heart muscle was described as opaque.

Case III (No. 36484). The third case was that of a young man of 22 with no history of previous illness excepting for an attack of typhoid fever five years before admission; on the eighth day of a typhoid fever of moderate intensity, a short, sharp murmur followed the second aortic sound and was transmitted downwards along the sternal margin. Five days later it was noted that the second sound was rather indefinite and "murmurish" at the aortic area, though no positive murmur was audible. A distinct systolic murmur was heard over the body of the heart and in the pulmonic area, fainter in the second right space. The patient made an uneventful recovery. No further note was made upon the heart.

Case IV (No. 41270). A man of 40, who had had rheumatism 23 years before, developed, on the 11th day of a mild typhoid fever, an apical systolic murmur, and on the 14th day, a diastolic, heard along the left sternal border, both of which cleared up before discharge. The apex, however, moved outward 2.5 cm.

In a very large number of cases in which a systolic murmur at the apex was not noted it is mentioned in the history that, at the height of the disease, the first sound was "murmurish," "feeble," "blurred," "indistinct" or "indefinite." Later, however, in all of these cases in which a note was made, the condition of the heart sounds was found to be normal.

In a number of instances reduplications of the first and second sounds were recorded but sufficient attention was not paid to the study of reduplications to justify any conclusions.

(3) Pericarditis.

There were but 2 instances of pericarditis among these 1458 cases.

Case V (No. 24135). The first case was that of a man of 20 who, on the 26th day of a rather severe typhoid fever, with pronounced nervous symptoms—delirium, subsultus, general tremor—showed, just outside the point of maximum impulse, a very distinct to-and-fro friction rub. For several days this increased in intensity and then gradually disappeared. The patient made an uneventful recovery.

Case VI (No. 30666). The second case was that of a colored man 23 years of age. Early on the morning of the 25th day of his disease, after a tub, the patient began to complain of pain in the præcordial area and in the right upper chest. A well-marked pericardial friction rub was audible, though no definite endocardial murmur could be made out. The patient grew rapidly weaker; an audible and palpable friction rub developed in the lower axilla and back, death following within twenty-four hours

The necropsy showed, beside the lesions of typhoid fever, a fibrino-purulent pericarditis, due to the softening of tuberculous bronchial lymph glands and perforation of the trachea, œsophagus and pericardium. The typhoid bacillus was obtained on culture from the pericardium, bile and spleen.

Case VII (No. 45535). A third case of pericarditis complicating typhoid fever is at present in the hospital.

The patient, a woman aged 29, entered the ward toward the end of the first week of the disease. Four days before she had been seized with a stabbing pain in the region of the heart and a sense of palpitation. On entrance there was a systolic murmur in the pulmonic area and a well-marked to-and-fro pericardial friction rub in the præcordial region. No pleural friction rub was audible. Several days later evidences of a patch of bronchopneumonia were to be detected in the left axilla. The cardiac dulness increased, and the heart sounds became more muffled. Gradually, however, in the course of two weeks, the friction murmur entirely disappeared. The cardiac dulness diminished, the areas of pulmonary consolidation cleared up, and at the present day the patient is in full convalescence.

(4) Endocarditis.

Three cases of endocarditis have been discovered at necropsy. None of these were recognized *intra vitam*. In addition, there were several cases in which endocarditis has been suspected clinically.

Case VIII (No. 10131). The first case has already been reported by Dr. Osler.[2] A. C., a colored girl of 20, was admitted to the hospital on the 7th day of her disease. Just before admission the patient had had several bloody stools, and within two hours after entry there were five more which consisted almost entirely of blood. The heart's action was rapid, the sounds clear. There was a suggestion of embryocardia. The patient's condition, which was very grave on entry, did not change for the better and she died on the following afternoon.

At the necropsy, beside the lesions of typhoid fever, it was noted that the heart was not enlarged. "All the valves were normal except the mitral. Along the edges of the leaflets of this valve are a number of translucent vegetations varying in size from the head of a pin to four or five times that bulk. These are comparatively recent. The heart muscle is dark, brownish-red and slightly mottled by patches of a lighter color." The aorta was normal, the coronary arteries normal.

Microscopically, the heart showed diffuse, fine, fatty degeneration. Typhoid bacilli were cultivated from the spleen, liver and kidneys. Staphylococcus albus was obtained from the endocarditic vegetations.

Case IX (No. 28194). M. S., a man of 18, was admitted to the hospital on the fifth day of a very severe typhoid fever. On entry the heart sounds were loud and clear. Delirium soon developed and the pulse became rapid and feeble. On the following day a rather soft systolic murmur was heard at the apex, transmitted to the axilla. The second aortic sound was markedly accentuated. The patient's condition did not change essentially; the delirium and picking at the bed clothes continued. The leucocytes, which, on admission, had been 6000 to the cm., were on the 7th day, 17,000 and 28,000, and on the following day 18,600. On the evening of the 8th day the patient died. At the necropsy, which revealed characteristic typhoid lesions, the following note was made upon the heart. "Along the edges of the mitral valve there is a row of minute dew-drop-like excrescences which are less than pin-head in size. These are fairly firm, not easily scraped off. They can, however, be pulled off with forceps. Smaller similar excrescences are found on the aortic segments. Otherwise the valves are delicate and apparently competent. The heart muscle is rather soft, natural in color. The vegetations on the mitral valve are on the auricular side. To those on the aortic valve there is attached a soft clot." The aorta showed slight fatty degeneration of the intima.

Cultures showed both the bacillus typhosus and staphylococcus aureus from the heart's blood; staphylococcus aureus from the vegetations on the aortic valve.

Case X (No. 44240). The third case, C. M., a man of 25, was admitted to the hospital in the first week of a very severe typhoid fever. He had always been strong, athletic and vigorous. He had had measles in childhood and bronchial asthma off and on for eleven years. He was very ill and delirious from the onset. The heart's action was rapid, the sounds, however, always clear. Four days after entry the first sound was reduplicated at the apex, the second at the base. Eleven days after entry a phlegmon developed in the left submaxillary region. This was opened on the following day, cultures showing staphylococcus aureus. Twelve days after entry the patient suddenly died.

Necropsy showed numerous small translucent vegetations along the line of closure of the anterior segment of the mitral valve. The heart muscle was rather soft, opaque and grey, with numerous more opaque, distinct grey spots. The anterior descending branch of the coronary artery showed many yellow flakes of sclerosis varying from 1 to 2 mm. in diameter. Almost the whole intima was converted into irregular, yellow grey patches which were slightly elevated. The walls of the smaller branches were also thickened. The aorta showed only slight thickening just above the valves. In the wall of the left ventricle, near the auriculo-ventricular ring, there were a number of small patches in which the muscle was completely replaced by greyish-white, somewhat translucent fibrous tissue. Cultures from the vegetations showed staphylococcus aureus and albus.

[2] Johns Hopkins Hospital Reports, 1895, V, 467.

It will be noted that the presence of endocarditis was unsuspected during life in all these cases. Indeed, there was nothing in the symptoms of the first or third cases which would have justified such a diagnosis. The development in the second case of the murmur, in association with a marked leucocytosis might have suggested the complication.

In three cases there developed during the disease, symptoms which persisted on discharge to an extent suggestive of an actual valvular lesion.

Case XI (No. 23799). The first case, a girl of 9, was admitted to the hospital on the 31st day of her disease. She had had no serious previous illnesses. The first sound on admission was nearly replaced by a soft blowing systolic murmur which was heard, though less intensely, as one passed toward the axilla; lost in the anterior axillary line. It was also audible over the base and præcordial region. The second pulmonic sound was somewhat accentuated. The apex was in the 4th left space, 6 cm. from the midsternal line. On the final note, over two months later, the point of maximum impulse was in the 4th space, 7 cm. from the median line, and the murmur, which was rough and rumbling, entirely covered and followed the first sound; it was, however, of greater intensity in the pulmonary area and was lost beyond the anterior axillary line. The leucocyte count varied between 6000 and 15,600.

It is of course possible that, in this case, the lesion may have preceded the typhoid fever, although there was no history of previous infections.

Case XII (No. 40588). The second case, M. S., a colored girl of 13, was admitted to the hospital on the tenth day of her disease. On entrance the heart sounds were clear at the apex and base. The leucocytes, which were 8800 on entry, varied during the next several days, between 6000 and 14,000. On the 19th day, without apparent cause, they were 18,600; on the 23rd day, 7500. No further note as to the condition of the heart was made until the time of discharge, the 66th day of the disease, the 14th of perfectly normal temperature. On this date there was heard, at the apex, a very faint systolic murmur, which was lost as the sternum was approached; it was, however, transmitted into the axilla as far as the mid-axillary line, and became intensified in the erect posture. The second pulmonic was accentuated. The pulse was 23 to the quarter, somewhat irregular in rhythm and volume, the irregularities mainly dependent on respiration.

The development and persistence in this case, of an apical systolic murmur in association with a rather unaccountable leucocytosis may be regarded as suspicious symptoms.

Case XIII (No. 44724). The third case was that of a man of 18 who had previously suffered from measles as a child and some severe illness, the nature of which he does not know, at four. He entered the hospital on the 9th day of a typhoid fever of moderate severity. On the 10th day the point of maximum cardiac impulse was in the 4th space 7 cm. from the mid-sternal line; the sounds were loud; the first, reduplicated at the apex and over the right ventricle. A well-marked systolic murmur was audible in the second and third interspaces in both aortic and pulmonic areas. It was not transmitted to the vessels of the neck, nor was any thrill to be felt in the aortic area. The

patient passed through a mild attack of typhoid fever with a ten day's relapse. The temperature, between the 40th and 60th days, was slightly irregular, occasionally rising above 99°. The colorless corpuscles, during the early part of the malady, ranged from 3 to 4000. On the 69th day the leucocytes were 3800. On the 79th day, while the temperature was subnormal, it was noted that the point of maximum impulse was 10 cm. from the mid-sternal line, 3 cm. farther out than on entry. A definite systolic thrill was felt all over the cardiac area. At the second rib on the left sternal margin there was a distinct systolic murmur. The leucocytes were 7800 to the cu. mm. Two days later the point of maximum impulse was in the 5th left interspace, and a blowing systolic murmur was audible all over the cardiac area. This was well heard in the pulmonic region but over the aortic area it had become intensified and rougher.

On the following day, I found, at the base and especially in the aortic area, a very loud echoing systolic murmur. This reached its maximum in the first right space where it was harsh and rasping. It was well heard in the epi-sternal notch, slightly in the carotids. After exercising and lying down again a soft systolic murmur was heard at the apex. The pulse was distinctly long and well sustained, the tracings showing characteristic flattening of the summit of the curve.

This case, which was seen also a month after discharge, presents evident signs of aortic stenosis.

It is interesting to note that at the time when the character of the murmur at the base began to change, the leucocytes which had been almost invariably under 4000, went up to 7800. It seems reasonable to assume that in this instance an aortic endocarditis developed during the typhoid fever.

(5) MYOCARDITIS.

There were few cases in which a definite diagnosis of acute myocarditis was made *intra vitam*. Indeed the symptoms upon which such a diagnosis may be based are still extremely questionable. Extreme rapidity and irregularity of the heart's action, embryocardia, the development of relative mitral insufficiency would lead one to suspect the existence of grave acute changes in the heart muscle. All these symptoms may, however, depend upon vaso-motor paralysis, indeed, the observations of Pässler and Rolly,[23] would tend to suggest that this is often the cause of the collapse at the height of many acute diseases, the true cardiac changes coming on in great part secondarily to and as a result of the impaired nutrition of the heart wall incident to the fall in blood pressure.

The heart muscle in many of the cases dying with symptoms of marked cardiac collapse showed the characteristic appearances described by the old observers—general flabbiness, a yellowish brown, dead leaf color, looseness, œdema and diminished consistency of the heart muscle and, not infrequently, a distinct mottling suggestive of fatty change. In how far the development of systolic apical murmurs at the height of typhoid fever may be regarded as indicative of true myocarditis is a point which we must regard at present as quite unsettled.

A comparison of the more minute histological changes in the heart wall with the clinical symptoms in our fatal cases is one of the important points of this investigation which I have not as yet been able to approach.

One case which must be regarded as an example of weakness of the heart muscle immediately subsequent to what was probably a mild typhoid fever may be of interest.

CASE IV[24] (No. 41270). F. J., aged 40, was admitted to the hospital on the 4th day of a mild typhoid fever. He had had rheumatism 23 years previously, and chills and fever three weeks before entry. On entry the heart sounds were clear though the first was rather muffled. On the 11th day a well-marked systolic murmur was heard at the apex, almost completely replacing the first sound and propagated to the anterior axillary line. It was not heard in the tricuspid area, nor above the 4th rib. At the base, both sounds were clear. Three days later it was noted that the pulmonic second sound was accentuated, while a soft diastolic murmur was heard along the left sternal border in addition to the systolic murmur at the apex. On the 18th day the temperature was subnormal, the diastolic murmur had entirely disappeared and the systolic murmur was barely audible. On the day of discharge, the 34th, the heart sounds were clear excepting a slight reduplication of the second in the pulmonic area. The apex impulse was, however, 11 cm. from the median line, 2.5 cm. further out than on entrance.

The fact that these cardiac manifestations were associated neither with fever nor with leucocytosis speaks rather against the existence of a true endocarditis and in favor of a purely myocardial weakness.

(6) VENOUS THROMBOSIS AND PHLEBITIS.

Among the 1458 cases of typhoid fever there were 38 instances of venous thrombosis, a percentage of over 2.6.

In addition to these cases two patients with this complication were admitted during convalescence, while one man who had suffered from double iliac thrombosis complicating typhoid fever entered the hospital two years later on account of the resulting symptoms.

Mortality.—5 or 13.1 per cent of these cases resulted fatally.

In two instances the thrombosis was probably the primary cause of death, once, through pulmonary embolism from a thrombus in the left axillary artery (No. 27855), once (No. 28,600), in a case of thrombosis of the left common iliac and femoral veins, through the lodging of a large embolus in the inferior cava and right auricle.

SITUATION OF THE THROMBUS.

(a) As to section of the body.

In these 41 cases the thrombosis was:
- In the lower extremities in 39 cases.
- In the upper extremity in 1 case.
- In the pulmonary artery in 1 case.

(b) As to side.

The occluded vessel was:
- On the left side in 26 instances.
- On the right side in 5 instances.
- On both sides in 8 instances.
- Imperfect record in 2 instances.

[23] Experimentelle Untersuchungen über Kreislaufstörungen bei acuten Infectionskrankheiten. Deutsch. Arch. f. klin. Med., 1903, LXXVII, 1.

[24] Vide supra, p. 327.

(c) As to vessels.

The femoral vein was occluded in 20 instances.
The popliteal vein was occluded in 5 instances.
The iliac vein was occluded in 5 instances.
The veins of the calf were occluded in 5 instances.
The internal saphenous was occluded in 3 instances.
The pulmonary artery was occluded in 1 instance.
The pulmonary artery and common iliac vein were occluded in 1 instance.
The axillary vein was occluded in 1 instance.

In six of the cases of femoral thrombosis, plugging of the internal saphenous was also noted.

Symptoms.

Time of onset.—The thrombosis occurred usually toward the latter part of the fever and in a number of instances after complete defervescence. The time of onset is illustrated in the following table:

Table III.

Illustrating the Time of Onset of 38 Cases of Venous Thrombosis.

	WEEKS.													
	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th	11th	12th	13th	?	Total
Cases	1	1	10	8	5	3	2	4	0	1	1	0	1	1	=38

Fever.—In 35 of our 38 cases the development of the thrombosis was associated with fever; in 3 there was no fever.

In 25 cases the fever was intercurrent, the phlebitis coming on before complete defervescence.

In 13, the temperature was normal at the time of the onset of the phlebitis.

Chills.—In 11 or 28.9 per cent of these 37 cases the complication was associated with chills.

In 2 instances the chills occurred just at the time of recognition of the condition.

Twice the chills occurred before recognition, once four days and once a week before.

In 6 cases the chills occurred during the course of the complication, once during the period of subsidence and in the other five instances during the height of the process.

Once, the chills occurred before, during and after the apparent onset of the thrombosis.

In one of the three cases entering the hospital after the acute symptoms had passed, there was a history of chills, their exact relation, however, to the time of development of the thrombosis was not clear.

Pain.—The first definite symptom of the thrombosis in every instance was pain. This varied considerably in intensity and suddenness of onset.

(a) Character of the first symptoms in 18 cases of femoral thrombosis.

In all the cases of femoral thrombosis but one there was pain along the course of the femoral vein. In this one instance the only symptoms noted in the hospital were those of popliteal thrombosis. The subsequent history of the case, however, made it clear that there must also have been plugging of the femoral vein.

The first localizing symptom of the process was:

Pain along the course of the femoral vessels and in Scarpa's triangle in 9 instances.
Pain in the calf in 4 instances.
Pain in the popliteal region in 1 instance.
No satisfactory note in 3 instances.
Patient too ill to localize pain in 1 instance.

In 3 of these cases the onset of the pain was strikingly sudden.

(b) Character of the first symptoms in 5 cases of popliteal thrombosis.

In all of the cases of popliteal thrombosis the first symptom was pain in the popliteal region.

(c) Character of the first symptoms in 5 cases of iliac thrombosis.

In the cases of iliac thrombosis the onset was associated with sudden sharp abdominal pain in 4 cases. In one of these instances the pain was extremely severe and associated with marked leucocytosis, the symptoms suggesting perforation. An exploratory laparotomy was performed, revealing the true nature of the lesion. In one instance in which the patient was hemiplegic and dull, there was no pain.

(d) Character of the first symptoms in 5 cases of thrombosis of deep veins of the calf.

In all of these cases the first symptom was pain in the calf.

(e) Character of the first symptoms in 3 cases of thrombosis of the internal saphenous vein.

In 3 cases in which there was thrombosis of the internal saphenous vein alone the first symptom was pain along the course of the vein in every instance.

(f) In the one case of axillary thrombosis the first complaint was of pain about the bend of the elbow.

Œdema.—In all of the 38 cases there was swelling of the affected part and in most instances the local temperature was raised. Often there was redness over the affected vessel. A definite note as to the existence of œdema was unfortunately not made in all cases.

(a) In 18 cases of femoral thrombosis:

Œdema was noted in 10 cases.
"Swelling" alone, in 6 cases.
There was no œdema in 2 cases.

(b) In 5 cases of iliac thrombosis there was:

Œdema in 2 cases.
No œdema in 3 cases.

(c) In 5 cases of popliteal thrombosis there was:

Œdema in 2 cases.
"Swelling" in 1 case.
No note in 1 case.
No œdema in 1 case.

(d) In 5 cases in which the veins of the calf were apparently involved alone there was:

Œdema in 1 case.
"Swelling" in 3 cases.
No œdema in 1 case.

(e) In all 3 cases in which the internal saphenous vein and calf muscles were involved there was œdema.

(f) In the case of thrombosis of the axillary artery there was œdema.

Palpability of the thrombotic mass.—In 16 out of 38 cases the thrombotic mass was felt as a palpable cord.

Leucocytosis.—In 21 cases counts of the colorless corpuscles were made during the complication.

In 12 or 57.1 per cent the leucocytes were above 10,000.

In 18 or 85.7 per cent they were above 7000.

In one instance they were below 6000.

The highest count, 24,864, occurred in a case of iliac thrombosis in the third week of the disease. In this instance an exploratory laparotomy was done in view of the possible existence of perforative peritonitis. The lowest count, 2700, occurred on the day after the onset of a phlebitis, apparently of the popliteal, which occurred in the sixth week without fever and with very slight general symptoms.

In connection with these blood counts it should be remembered that, in every instance but one the phlebitis came on in the third week of the disease or later, at a period when, according to our statistics, the leucocyte count in uncomplicated cases should not be above 5500.

Association with secondary infections.—Out of our 38 cases there were 7 instances in which secondary infections preceded or co-existed with the onset of the thrombosis. These secondary processes were:

Boils in 2 cases.
Well-marked bronchitis in 1 case.
Gangrene of the lung in 1 case.
Fistula in ano in 1 case.
Chronic otitis media in 1 case.
Amœbic dysentery with developing hepatic abscess in 1 case.

In the remaining 31 instances there was no clinical evidence of secondary infection.

Post-mortem appearances.—In the 3 cases in which bits of the clogged vein were examined histologically, evidence of well marked inflammatory changes were found in the walls of the vessels, such, however, as might well have arisen secondarily to the thrombosis. In one of these cases there was at the point of section, an extensive endophlebitic plaque which however, probably antedated the typhoid fever. But few sections were examined and it is improbable that these came from the point of origin of the thrombi, so that conclusions with regard to the pre-existence of a phlebitis are not justified.

Cultures.—In one case the bacillus typhosus was obtained in pure culture from the thrombus.

In 1 the cultures were negative.

No notes were made with regard to cultures from the thrombus in the other 2 cases.

A consideration of the character and extent of the general symptoms in this group of cases would seem to justify the conclusion that in most instances the thrombosis was associated with and preceded by a phlebitis.

Especially interesting is the frequency of chills in association with the complication. Unaccountable chills at the height or toward the latter part of typhoid fever, should always give rise to the suspicion that a phlebitis may be developing.

(7) Arterial Thrombosis and Arteritis.

Five cases of arterial thrombosis or arteritis developed under our observation.

CASE XIV[35] (No. 12616). The first case was that of a young man of 22, of excellent family and personal history, who was admitted to the hospital on the 24th of April, 1895, on the 4th day of a mild typhoid fever. On the 9th day he was seized suddenly with violent general convulsions, the movements being rather more marked on the right side. There was conjugate deviation of the eyes upward and to the left. The convulsions recurred at short intervals for about an hour. During the paroxysms, the patient was profoundly unconscious, though between times his mind seemed partially clear. After an intermission of about four hours the convulsions returned, recurring with great severity at intervals for six hours, when death occurred during a severe paroxysm.

The necropsy performed by Dr. Flexner, showed a thrombosis associated with an acute arteritis of the ascending parietal and parieto-temporal branches of the left middle cerebral artery. The extent of the arteritis, together with the fact that it was found in branches in which there was as yet, no thrombosis, led us to regard it as having preceded the occlusion of the vessel.

CASE XV[36] (No. 28600). The second case was that of a man of 46 who entered the Johns Hopkins Hospital on the 27th of November, 1899, on the second day of typhoid fever. He had had measles and mumps as a child and a year before a slight arteritis of the left ankle. He had had a venereal ulcer of doubtful character, without secondary symptoms, seven or eight years before. On the 19th day of his disease, while delirious, he gradually developed a complete hemiplegia of the left side. The respiration became of a Cheyne-Stokes character, which persisted during the next several days. There was no loss of consciousness. Five days later he died suddenly from embolism of the inferior cava, resulting from the breaking off of a large thrombus in the left iliac vein. The necropsy showed an area of softening in the upper part of the right internal capsule, due undoubtedly to the plugging of the branch of the artery supplying the area. The leucocytes, which on entry, were 6200, were 9250 to the cu. mm. during the onset of the symptoms of occlusion.

CASE XVI[37] (No. 39264). The third case was that of a girl of 16 who entered the hospital on the 16th of June, 1902, on the 5th day of a severe typhoid fever. There was a marked general bronchitis, and on the 8th day of her illness, evidences of bronchopneumonia appeared. The leucocytes at this time were, on two counts, 6000 and 5000 to the cu. mm. On the 11th day there was a slight leucocytosis. On the succeeding three days the patient seemed better until, on the 14th day, on removing her from the tub, the right foot and leg below the knee were found to be white and cooler than on the left side. The coolness extended above the knee in front. There was no swelling of the leg or foot. The femoral artery could be followed by its pulsation half way down the thigh. No pulse was palpable in the

[35] Reported by Dr. Osler, Johns Hopkins Hosp. Reports, 1900, VIII, 364; and by Thayer, N. Y. State Journal of Medicine, 1903, III, 21-28.

[36] Reported by Dr. Osler. Johns Hopkins Hosp. Reports, 1900, VIII, 368.

[37] Reported by Thayer. N. Y. State Journal of Medicine, 1903, III, 21-28.

popliteal or dorsalis pedis. The patient was delirious and in a condition of profound intoxication; the leucocytes were 17,000 to the cu. mm. Gradually, a line of demarkation developed about at the junction of the upper and middle thirds of the leg with dry gangrene below. The condition of the patient became gradually worse and death followed on the 22nd day. The heart's action was rapid throughout the disease, but there was no evidence of dilatation, the sounds were always clear, and there were no signs of embolism elsewhere. The case was regarded by Dr. McCrae and the writer as one of thrombosis rather than embolism. No necropsy was allowed.

CASE XVII[28] (No. 39471). The fourth case was that of a boy of 18 who, on the 14th day of a fairly severe typhoid fever, began to complain of pain and tenderness on the inner side of the thigh along the course of the femoral vessels, extending subsequently to Scarpa's triangle. The left thigh was warmer than the right and swollen, while the foot and leg were pale and cool. There was no œdema. The pulse in the femoral and arteries of the foot almost disappeared. The symptoms soon began to improve, but on the 49th day, pain and swelling appeared in the popliteal space and calf, the foot became cooler and cyanotic again, and the arterial pulse in the foot once more entirely disappeared. There was slight œdema about the malleoli. The œdema lasted but a few days; the pulsations in the arteries gradually improved, and the patient was discharged apparently well on the 71st day.

CASE XVIII[29] (No. 40343). The fifth case was that of a woman of 27, who on the 21st day of a mild typhoid fever, began to complain of pain at the bend of the elbow where there was marked tenderness with slight redness and swelling. The radial pulse was diminished in size and the hand cooler and paler than the right. In the course of several days, however, the symptoms wholly cleared up.

In these two latter cases it is a question whether an actual thrombosis developed or whether the symptoms may not be regarded as due to an acute arteritis alone, the interference with the circulation following the œdema and infiltration of the walls.

In addition to these cases there have been treated in the hospital 3 instances of hemiplegia in which the onset occurred during typhoid fever, while Dr. Thomas has kindly given me the notes of two further cases which he has observed in the out-patient department.

CASE XIX[30] (No. 14083). The first case was that of a girl of 7, who entered the hospital on October 3, 1895, complaining of inability to use the right hand. On June 3, during the 9th week of a rather severe attack of typhoid fever, the child was seized with violent convulsions confined to the head, right arm and leg. On the afternoon of the day of onset the movements of the head, which the mother described as having been thrown backward, ceased. Continued movements of flexion and extension of the limbs followed for two days. When these came to an end, it was noticed that the right arm and leg were paralyzed. There was also involvement of the face and total loss of speech. Five weeks after onset spastic rigidity of the arm was noticed. Six weeks after the onset movements of the leg began to return. In the seventh week there was some return of speech. At the time the patient was in the hospital, the speech was improving rapidly, though it was still very imperfect. The child recognized objects and looked bright and intelligent. The right leg was dragged, the foot inverted. The right arm was held semiflexed, the wrist and hand flexed with marked spastic rigidity. She could voluntarily flex and extend the arm at the elbow and could lift the hand to the head, but the power of extension in the wrist and of the fingers and the ability to grasp objects with the hand were almost completely lost.

CASE XX[31] (No. 14630). The second case was that of a man of 25, of excellent family history, who entered the hospital on November 30, 1895. In March, at the end of the second week of a severe attack of typhoid fever, a paralysis of the left arm and leg suddenly developed. There were no convulsions, and his physician states that there was no aggravation of the delirium during the attack. There was no difficulty in speaking and no trouble with the rectum or bladder. When in the hospital there was no trace of paralysis of the facial muscles; the left arm could be moved at the shoulder and elbow and slightly at the wrist in flexion. The hand could be extended but only slight movements of extension of the fingers were possible. The power of pronation and supination was lost. The left leg could be moved freely at the thigh and flexed and extended at the knee. The reflexes on the left were everywhere increased. Wide, irregular, choreiform movements were noted when the patient attempted to move the left arm.

CASE XXI (No. 30540). The third case was that of a man of 34, who was admitted to the hospital on the 19th of May, 1900. In the fall of 1897 he had had a severe typhoid fever during the third week of which he became delirious. On recovering consciousness, in the 9th or 10th week, he found that his left arm and leg were paralyzed. Three years later, at the time he was in the hospital, he showed distinct loss of power, with spastic contractions in both extremities and athetoid movements of the hands.

CASE XXII (Nervous Dispensary No. 13149). The fourth case was that of a girl of 8, who entered the Nervous Department of the Dispensary on the 13th of March, 1902, complaining of weakness on the right side. There was nothing remarkable in her family or previous history. Four and a half years before, in October, during the third or fourth week of a typhoid fever, she developed a right-sided hemiplegia. There were no violent convulsions though it is stated that "she straightened out." She was unable to clearly understand what was said to her. There was loss of control of the bladder and rectum, and involvement of the facial muscles. The power of speech began to return about eight months after the onset; she was unable to walk for a year. The facial paralysis disappeared after several months and the control of the bladder and rectum after about six months. At the time when she reported at the dispensary, the right arm was wasted and shorter than the left. There was wrist drop, and the fingers were flexed; there was spastic contraction of the flexors at the elbow and stiffness on trying to extend the wrist. There was slight toe drop on the right side, though the patient walked fairly well, the right foot being dragged but little.

CASE XXIII (Nervous Dispensary No. 15179). The fifth case was that of a girl of 10, of good family and previous history excepting for the fact that at 2 she had had chills and fever and convulsions. At the age of 4 she had typhoid fever. After about three weeks she was so much better that she sat up in bed. This was followed by a relapse in which there developed a right-sided hemiplegia. While sleeping, she suddenly waked with a scream and fell back unconscious. There were no convulsions. She did

[28] Reported by Thayer. N. Y. State Journal of Medicine, 1903, III, 21-28.

[29] Reported by Thayer. N. Y. State Journal of Medicine, 1903, III, 21-28.

[30] Reported by Blumer. Johns Hopkins Hospital Bulletin, 1896, VII, 72.

[31] Reported by Dr. Osler. Johns Hopkins Hospital Reports, 1900, VIII, 367.

not recognize any one for 21 days, and did not walk for a year. Speech began to return after about three months. At the time when she consulted Dr. Thomas, six years after her illness, there were contractures and athetoid movements in both arm and leg.

In this connection I may mention a very remarkable instance which was communicated to me a year ago by Dr. Royster of Norfolk.

Case XXIV. A girl of 8, in the 4th week of a typhoid fever of moderate severity, when the temperature had nearly reached a normal point, developed a left-sided hemiplegia involving the face, arm and leg, in association with paralysis of the right third nerve—a typical Weber's syndrome. The onset was associated with convulsions. A year later the child still had at times spasmodic twitchings involving the left side and the right eye and occasional general convulsions. The lesion in this case was undoubtedly peduncular, probably thrombotic.

Two cases of cerebral thrombosis with aphasia and hemiplegia which I have previously reported,[33] may be also included in this list. Both of these instances were observed in the Massachusetts General Hospital.

Case XXV. The first of these cases was that of a man of 21, of good family and previous history, who on the 10th day of a fairly severe typhoid fever, developed a complete right-sided hemiplegia with aphasia. There were no convulsions; the patient was dull but not unconscious at the time of onset. About three months later he was able to walk and the power of speech was returning. There was still, however, but little return of power in the forearm and hand.

Case XXVI. The second instance was that of a girl of 10, who, in the 4th week of typhoid fever, developed paralysis of the right arm with aphasia. At the time of onset the child was in a dull stupid condition but there were no convulsions and no loss of consciousness. Six weeks later, the movements of the hand and arm were almost as good as on the other side, but the child seemed mentally dull.

I have also the notes of an extremely interesting case, the anatomical specimens from which were sent to me by my friend Dr. Akerman of Wilmington, North Carolina.

Case XXVII. The patient, a man of 31, of good habits, with no history of previous serious illnesses, entered the James Walker Memorial Hospital on July 1, 1903, on the third or fourth day of a severe typhoid fever. At the end of the second week (13th or 14th day) there developed a complete left-sided hemiplegia. The patient was delirious at the time of onset of the paralysis but not unconscious. Two weeks later, despite the gravity of the condition, signs of return of power on the paralyzed side began to appear. In the beginning of the 5th week (July 25) there developed a right femoral phlebitis. The patient, who was very ill throughout, gradually failed and died on the 41st day. The autopsy showed extensive infarctions of the spleen and kidneys, due apparently to autochthonous thrombosis. The heart showed nothing remarkable; the brain, which was preserved whole, was unfortunately lost on account of a mistake in the preserving fluid, so that the exact location of the lesion could not be determined. There can, however, be little doubt from the character of the case that it was an instance of thrombosis.

Time of Onset of Symptoms.

Considering these 14 cases together the onset of the symptoms occurred:

In the second week in 4 cases.
In the third week in 4 cases.
In the fourth week in 3 cases.
In the third or fourth week in 1 case.
In the sixth week in 1 case.
In the tenth week in 1 case.

Of the 8 cases in which the onset was observed, in all but one it occurred during the febrile period.

The complication appeared to have slight influence on the course of the *fever*, excepting in the first case where the temperature rose up to the time of death.

In none of the cases were there *chills*.

Leucocytosis.—In the four cases in which blood counts were made the leucocytes were: 17,000
13,200
9,500
9,250

Taking into consideration the clinical symptoms in connection with the temperature records and the microscopical observations in the first case, it would seem not improbable that in all of these cases there was a primary arteritis. Whether in cases XVIII and XIX a partial parietal thrombosis occurred or whether the symptoms are to be ascribed to the interference with the circulation incident to the swelling of the arterial walls alone must remain an open question.

A striking point of variance between these cases and those of phlebitis is the difference in their distribution. Of the fourteen instances of arterial thrombosis but 2 occurred in the lower extremities. One developed in the arm and the remaining in the cerebral vessels.

Again, among these 14 cases the distribution as to the side of the body was more nearly equal, 6 being right-sided and 8 left-sided lesions. These particular figures cannot of course be compared with the cases of phlebitis which were in such preponderance, limited to the lower extremities. The statistics of Keen,[34] however, show that arterial thromboses are much more evenly distributed between the two sides.

Acute Aortitis.—We have never observed during life symptoms similar to those described by Potain,[35] as suggestive of the existence of acute aortitis, nor have our necropsy records shown changes which could well produce such symptoms.

III. Analysis of the Necropsy Records.

Between the fifteenth of May, 1899, and the first of January, 1903, there were 132 fatal cases of typhoid fever with 95 necropsies.

Heart Muscle.—Upon the gross appearances of the heart muscles comment has already been made. A study of the microscopical changes with modern methods of investigation, in those cases from which tissues have been saved, and a comparison of the results with the clinical histories is an import-

[33] Johns Hopkins Hosp. Bull., 1896, VII, 73.

[34] Surgical Complications and Sequels of Typhoid Fever. Philadelphia, 8°, 1898, 76-78.

[35] De l'aortite typhique. Semaine méd., Par., 1894, XIV, 460.

ant part of this investigation which I have as yet been unable to carry out. From what observations I have made I am convinced that there is room for further study of the changes occurring in the heart muscle in typhoid fever.

It is unnecessary here to refer to the fatal cases of acute endocarditis, pericarditis, phlebitis and arteritis, mention of which has already been made.

The Arteries.—Of especial interest, it seems to me, is the question as to the relation of acute infections in general and typhoid fever in particular to acute and chronic changes in the aorta and general arterial system. The occasional existence of acute arteritis in the larger peripheral vessels has already been mentioned in connection with arterial thrombosis. There is a feeling among many observers that the severe acute infections play a more or less important part in the causation of focal endarteritic changes in the aorta and other vessels which may subsequently give rise to grave functional disturbances, yet a really systematic research into the effects of typhoid fever upon the heart and arteries has not, so far as I know, been made.

A mere survey of the protocols of our necropsies reveals some interesting points. In a large proportion of these cases, unfortunately, little attention has been paid to the vessels, and where observations have been made, these are restricted almost entirely to the aorta and coronary arteries. In a considerable number of cases, however, especially in recent years, since our attention has been more particularly directed toward this point, relatively fresh sclerotic changes have been described in the intima of the aorta and coronary arteries.

The Aorta.—Out of 52 cases in which notes upon the condition of the aorta were made, there were evidences of sclerosis in 30. These changes were apparently recent in at least 21 instances.

The Coronary Arteries.—Especially interesting are the records with regard to the coronary vessels. Out of 62 cases in which the condition of the coronary arteries was recorded, there were 19 in which definite sclerotic changes were noted. In four other cases yellow opacities in the intima were described. In 13 of these cases the changes were early and apparently recent. These observations are, it seems to me, of considerable significance. They are markedly at variance with the above quoted experience of Curschmann.

One case, of recent occurrence, is particularly suggestive.

Case XXVIII (No. 40831). A strong, vigorous man of 25, of excellent history and habits, who had suffered from measles at 21, mumps at 23 and chicken pox at 24, died at the end of the third week of a very severe typhoid fever. The heart's action was rapid but there was no apparent dilatation and no cardiac murmurs. The pulse tension was remarkably low. At necropsy several very early patches of endarteritis were found at the root of the aorta, and one or two on the ventricular surface of the mitral valve; these were slightly raised areas, in part somewhat yellowish, in part still translucent. On opening the coronaries there were found "numerous patches of yellowish sclerosis, especially in the anterior descending branch. The posterior branch also shows extensive sclerosis. The patches are small and discrete and in large part translucent, spotted with yellow." Sections through these areas show a very early process, so much so that one may well ask whether the lesions did not originate in the course of the typhoid infection.

This patient was one of the house physicians, with regard to whose habits and life we had peculiarly good information. He was a man of exemplary habits who had not been given to excesses of any sort; he had never been in the habit of taking over-violent physical exercise, and as has been said, had suffered only from measles, mumps and chicken-pox. The lesions present in the coronary vessels were really extensive, and in the course of the regressive changes which must have followed had he recovered, might well have given rise to sufficient stenosis to result in the gravest damage to the cardiac muscle.

Such a case can but impress one with the importance of more careful observations upon this point. As Brault[*] said some years ago, "Alterations in the arteries of patients dead of typhoid fever should be carefully looked for, as well in the arteries of the extremities as in the aorta and arteries of the base of the brain. It is the only way to settle the question as to whether arterial affections are frequent in this disease."

IV. A Study of the Subsequent Condition of 189 Patients who passed through Their Typhoid Fever in the Wards of the Johns Hopkins Hospital within the Past Fourteen Years.

A point of fundamental importance with regard to the question of the effect of typhoid fever on the heart and vessels, would be the study of the subsequent condition of the vascular apparatus of a sufficient number of patients at a later period. The only method of solving this question would seem to be the careful observation of a large number of individuals during months and years following their typhoid fever. This is naturally a difficult thing to do, and the results of such an investigation must very naturally be affected by a variety of disturbing influences. Nevertheless it seemed to me that it might not be uninteresting to examine the hearts and arteries of as many of our old typhoid patients as could be reached, and especially, to observe the subsequent course pursued by those cases in which cardiac and vascular lesions were recognized at the time of their disease. Within the last two years I have examined 189 of our old typhoid patients. The results of these investigations, which have, in great part, been reported in the March number of the American Journal of the Medical Sciences[**] I will give here in brief:

(1) Age and Date of Attack.

The ages of the patients varied between 3 and 69 years, while the periods which had elapsed between the discharge from the hospital and the subsequent examination ranged from one month to thirteen years.

[*] Les artérites. Leur rôle en pathologie. Paris (not dated). Masson & Gauthier-Villars (Encyclopédie scientifique des aide-mémoire).

[**] On the Late Effects of Typhoid Fever on the Heart and Vessels. Amer. Journ. Med. Sc., 1904, CXXVII, 391.

The following table will show the period of time which had elapsed between the discharge of the patient from the hospital and the subsequent examination.

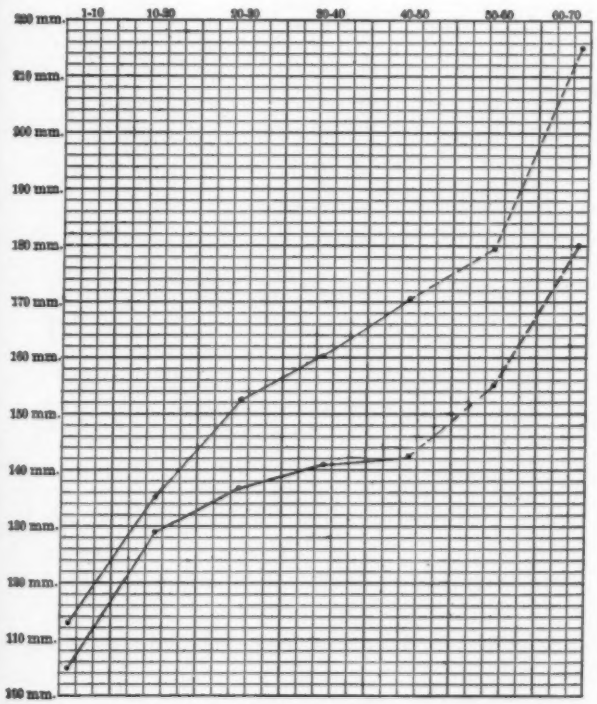
TABLE IV.

Showing the Length of Time which had Elapsed Between the Discharge of the Patient from the Hospital and his Subsequent Examination.

	MONTHS.						YEARS.							
	1–6[37]	6–12.	1	2	3	4	5	6	7	8	9	10	11	12	13
Cases	22	26.	19	21	23	22	11	13	11	10	6	2	1	1	1 = 189.

(2) *Pulse.*—With regard to the rate and regularity of the pulse, nothing especially remarkable was noted.

CHART I.



(3) SYSTOLIC BLOOD PRESSURE.

Of especial interest, however, were the observations upon the systolic blood pressure. These were made by means of the Riva Rocci apparatus. The patients were always in the recumbent posture, the estimate being made as the last step in the examination in order that psychical influences might be excluded as far as possible. The results of these estimations compared with a series of similar observations on healthy individuals who had never had typhoid fever are indicated in the following table which is graphically represented on Chart No. 1.

[37] There were but three cases seen under three months from the time of discharge.

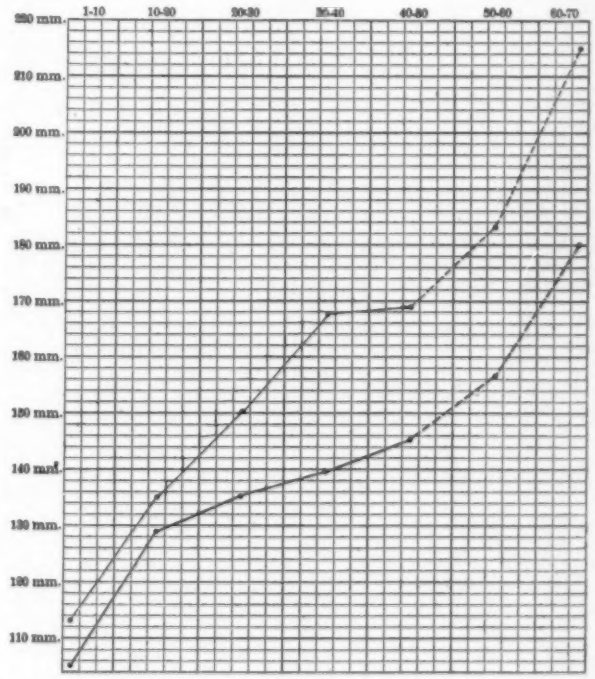
TABLE V.

Showing the Averages of the Systolic Blood Pressure in 276 Healthy Individuals and 172 Old Typhoids, Arranged According to Age by Decades.

	1-10	10-20	20-30	30-40	40-50	50-60	60-70
OLD TYPHOIDS.	112.4 mm. (5 cases).	135.2 mm. (39 cases).	152.5 mm. (61 cases).	160.8 mm. (48 cases).	170.2 mm. (15 cases).	179.6 mm. (3 cases).	215 mm. (1 case).
HEALTHY INDIVIDUALS.	104.6 mm. (37 cases).	128.7 mm. (87 cases).	136.9 mm. (89 cases).	140.8 mm. (37 cases).	142.2 mm. (20 cases).	154.8 mm. (5 cases).	180 mm. (1 case).

This table reveals the interesting fact that the average blood pressure in the old typhoids was uniformly higher than that in healthy individuals who had never had the disease. A

CHART II.



further analysis of our records shows that a large proportion of the old typhoids showed blood pressures above what are usually regarded as the normal limits.

In order to eliminate some of the numerous sources of error I prepared other tables excluding from each list all those cases in which a history of scarlet fever, diphtheria, acute rheumatism, pneumonia, erysipelas, small-pox or alcoholic habits could be obtained.

TABLE VI.

Showing the Averages of the Blood Pressure in Old Typhoids and Normal Individuals, from which all Cases with a History of Serious Infectious Disease or Alcoholic Habits have been Excluded.

	1-10	10-20	20-30	30-40	40-50	50-60	60-70
OLD TYPHOIDS.	113.2 mm. (4 cases).	134.9 mm. (28 cases).	150.3 mm. (34 cases).	167.6 mm. (27 cases).	168.7 mm. (8 cases).	183.5 mm. (2 cases).	215 mm. (1 case).

HEALTHY INDIVIDUALS.

105.5 mm. 128.3 mm. 135.1 mm. 139.8 mm. 145.2 mm. 156.4 mm. 180 mm.
(32 cases). (62 cases). (52 cases). (10 cases). (11 cases). (4 cases). (1 case).

These tables illustrated by Chart II, show, as may be immediately observed, no essential variation from the previous curve. The alteration of the typhoid curve, due to the higher figures in the column for the thirtieth and fortieth decades, only emphasizes the fact that the number of cases was still too small to allow of the construction of final charts.

(4) Palpability of the Radial Arteries.

Another striking feature in the examination of our old typhoids was the remarkable frequency with which the radial arteries were palpable. The following table which is illustrated by Chart III, shows a comparison of the figures in our old typhoids arranged according to decades, and a series of 421 healthy individuals who had never had typhoid fever. From our figures it appears that between the ages of ten and fifty years 48.3 per cent of the old typhoids showed palpable vessels, as compared with 17.5 per cent of the control cases.

CHART III.

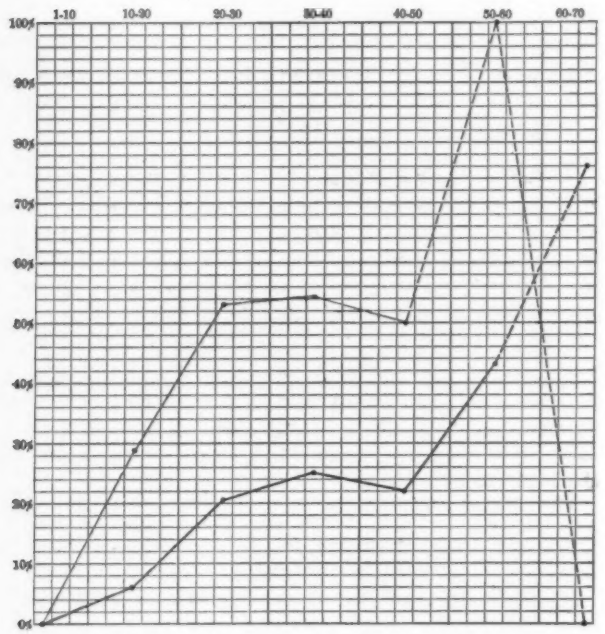


TABLE VII.

Showing the Percentages of Palpability of the Radial Arteries in 188 Old Typhoids and in 421 Healthy Individuals who had Never had Typhoid Fever, Arranged According to Age by

OLD TYPHOIDS.

1-10	10-20	20-30	30-40	40-50	50-60	60-70
0%	28.5%	53.1%	54.2%	50%	100%	0%
(5 cases).	(42 cases).	(64 cases).	(59 cases).	(14 cases).	(3 cases).	(1 case).

HEALTHY INDIVIDUALS.

0%	6.1%	20.4%	25%	22%	42.8%	80%
(37 cases).	(98 cases).	(186 cases).	(61 cases).	(27 cases).	(7 cases).	(5 cases).

As in the case of the blood pressure I have also prepared a second table including those cases only, in which a history of alcoholic habits and the more serious acute infections were absent. These figures, illustrated by Chart IV, show little variation from those based upon a larger number of cases.

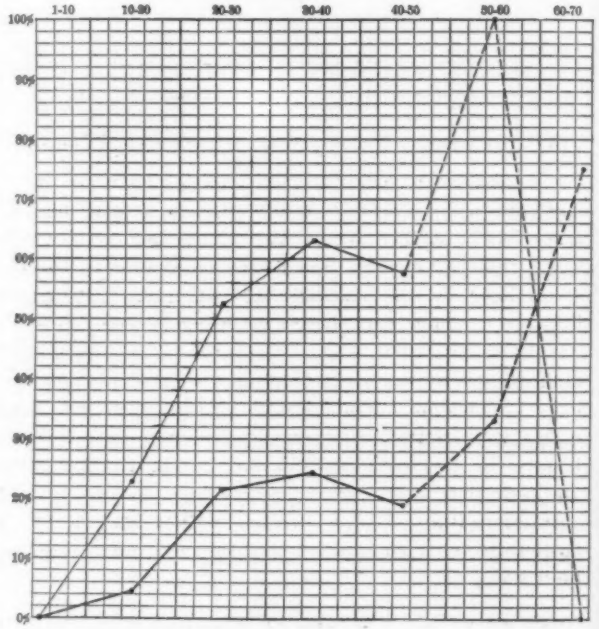
TABLE VIII.

Showing the Percentages of Palpability of the Radial Arteries in Old Typhoids and in Healthy Individuals from which All Cases giving a History of Serious Infections or Alcoholism have been Excluded.

OLD TYPHOIDS.

1-10	10-20	20-30	30-40	40-50	50-60	60-70
0%	22.5%	52.6%	63.3%	57.1%	100%	0%
(4 cases).	(31 cases).	(37 cases).	(30 cases).	(7 cases).	(2 cases).	(1 case).

CHART IV.



HEALTHY INDIVIDUALS.

0%	4.2%	21.3%	22.2%	18.7%	33.3%	75%
(32 cases)	(70 cases).	(103 cases).	(27 cases).	(16 cases).	(6 cases).	(4 cases).

Results so striking and constant as these could hardly be accidental, and interesting confirmation is afforded by the following observations.

In connection with some studies on albuminuria which are being made by Dr. Emerson, abstracts were made of 2000 consecutive histories in the Medical Department of the Johns Hopkins Hospital. Out of these 2000 histories a definite note as to the palpability or non-palpability of the peripheral vessels was made in 1174 cases.

In 943 cases in which the radial arteries were palpable 188 or 19.9 per cent gave a history of typhoid fever.

Out of 231 cases in which the peripheral vessels were not palpable but 25 or 10.8 per cent had had the disease.

The frequency of a history of typhoid fever in the two groups arranged by decade is illustrated by the following table.

TABLE IX.

Showing the Relative Frequency of a History of Typhoid Fever in 1174 Consecutive Cases Arranged According to Age by Decades.

Cases in which the peripheral vessels were palpable:

1-10	10-20	20-30	30-40	40-50	50-60
0%	6.35%	17.28%	17.94%	24.74%	22.35%
(2 cases).	(63 cases).	(191 cases).	(223 cases).	(190 cases).	(170 cases).

60-70	70-80	80-90
24.36%	29.17%	0%
(78 cases).	(24 cases).	(2 cases).

Cases in which the vessel wall was not palpable:

1-10	10-20	20-30	30-40	40-50	50-60	60-70
0%	11.76%	10.45%	8.77%	17.39%	14.28%	14.28%
(11 cases).	(51 cases).	(67 cases).	(57 cases).	(23 cases).	(14 cases).	(7 cases).

70-80
0%
(1 case).

These figures can but be regarded as strongly supporting the indications resulting from our previous observations.

(5) Condition of the Heart in 188 Old Typhoids.

A consideration of the condition of the heart in our old cases both in the hospital and subsequently, reveals the interesting fact that the average size of the heart was greater among the old typhoids than in the same cases at the time of admission to the hospital, the difference holding good also when the cases were classed according to age by decade.

In 12 of these cases, where, on discharge from the hospital, the heart was considered normal, subsequent examination revealed evidences of organic disease; in eight instances, hypertrophy with mitral insufficiency; in one, possible mitral stenosis; in one, aortic insufficiency; in one, marked general arterio-sclerosis with hyper-tension.[28]

An interesting case of hypertrophy with mitral insufficiency consecutive to typhoid fever has recently come under my observation.

Case XXIX. H. C. M., aged 33, a patient of Dr. Frank M. Sharpe, of Brooklyn, N. Y., consulted me on the second of March 1904. He had had measles as a child and pneumonia at the age of 4 or 5, but no other serious illnesses. His habits were excellent. He had always been a strong, athletic man. Two years ago last January, he had an attack of typhoid fever, during which he was in bed three or four weeks. Ever since this illness, he has been rather nervous, so much so that he felt obliged to give up his indoor work. He has noticed also that he is losing the "strength and the go" that he used to have, and he has been conscious of shortness of breath on going up hills. A month and a half ago he had a mild attack of influenza. About a month ago, on being examined for life insurance, he was told that he had cardiac trouble. The doctor also advised him to give up base ball and other out-of-door sports.

On examination, the pulse was slow, the artery of rather large size; duration moderate; slight dicrotism. Maximum pressure (Riva Rocci), 185. The radials were palpable on both sides; temporals not prominent. The point of maximum cardiac impulse was localized in the 4th space, 9.2 cm. from the median line, the dulness extending 4½ cm. to the right; the greatest diagonal dulness, right to left, was 15 cm. In the erect posture, the impulse, which was strong, was in the 5th space, 9½ cm. from the median line—after a little exercise, 10 cm. The first sound was prolonged and followed by a very slight systolic murmur which was lost two fingers' breadths outside the point of maximum impulse; it was not heard in the tricuspid area, but was just audible in both areas at the base. In the erect posture, however, the murmur increased in intensity and was faintly heard in the axilla and back, as well as all over the cardiac area. The second sound was louder in the aortic than in the pulmonic area. After exercise (dancing on one foot), the pulse was raised to a little over 100, but soon fell.

A study of the subsequent condition of those cases showing a remarkably rapid or irregular pulse during the disease revealed little of interest excepting for the fact that the blood pressure was somewhat above the common average for the old typhoids.

Subsequent History of 31 Cases of Typhoid Fever in which a Systolic Murmur at the Apex was Audible During the Attack.

The patients who, during their attack, developed a systolic murmur at the apex of the heart, showed a higher average blood pressure than the general mean of the old typhoids. In 28 of these cases, where the systolic pressure was taken on later examination, the mean was 158.5 mm. against a general average for the old typhoids of 152.4. This higher average was observed in every decade excepting the first in which there was but one case, and in the third, in which there were but four. In 18 cases over 20 years of age, the average pressure was 169.2 as compared with a general average of 159.6 for the same decades. In 5 cases the pressure was above 200 mm.

The average size of the heart was found to be greater than in the same cases at the time of admission to the hospital or in the old typhoids as a whole.

In 11 of these instances the heart sounds were clear on the later examination. In 20, murmurs were heard.

In one instance the murmur was a slight cardio-respiratory systolic sound audible at the apex only.

In 6 cases there was a soft systolic murmur at the base, in some instances heard also over the right ventricle.

In 13 cases there was a systolic murmur at the apex of the heart. In 5 of these cases the signs justified the diagnosis of mitral insufficiency.

In one case there were signs suggestive of mitral stenosis.

In one case there was a marked arterio-sclerosis with hypertension.

One case was an instance of Graves' disease with a systolic murmur all over the cardiac area. This condition was also present at the time of the fever.

From this, the striking fact appears that over one-fifth of those cases in whom during typhoid fever, an apical systolic murmur was audible, showed on later examination evidence suggestive of organic cardiac lesion.

[28] The full records of these cases may be found in Amer. Journ. Med. Sc., 1904, CXXVII, 410-420.

(6) Subsequent History of 16 Cases of Phlebitis and Venous Thrombosis.

Sixteen of our patients with venous thrombosis were seen or communicated with at a period of time, months or years after the onset of the complication. Ten of these cases were instances of femoral thrombosis; two, of double iliac thrombosis; two, of popliteal thrombosis; two, of thrombosis of the veins of the calf.

After history of 10 cases of femoral thrombosis.—One of these two cases was seen two and a half months after convalescence, one, ten days after his discharge from the hospital, while two others were communicated with years after the onset by letter. The remaining six were examined at periods varying from two to twelve years after the affection.

In all instances there was more or less permanent disability.

In 6 cases there had been *œdema* lasting for from several weeks to two years.

In 5 cases there was a complaint of *cramps* in the calves, especially at night and after exercise.

In one instance the patient complained of sharp *pains* running up through the leg and thigh especially after standing.

In one case the patient says that when he exercises his leg too much, "it becomes so weak that I can hardly use it; it seems to give out."

In every instance the affected leg and thigh were materially and permanently larger than the other.

In all cases, excepting in the instance seen ten days after discharge, there were marked *varicosities.* These were especially extensive in the calf, the popliteal space and on the posterior and inner side of the thigh. In 6 cases, all but one in which the condition was looked for, there was a well marked varicosity of the veins in the hypogastrium. This picture seems to be characteristic. The varicosities usually form a triangle with the apex a little below the umbilicus, the blood flowing from the affected thigh to the apex of the triangle and then down again into the opposite inguinal ring passing upwards evidently through the iliac vein of the other side. The one case in which these varicosities were missed was that of an ignorant colored man who was communicated with by letter.

After history of two cases of double iliac thrombosis. Two cases of double iliac thrombosis were seen several years after the onset of the complication. In one of these, the first symptoms of which had been abdominal pain with marked leucocytosis and the early appearance of rather prominent veins in the lower abdomen, the later, subjective symptoms were few. Extensive varicosities, however, developed in the veins of both legs and thighs, extending upwards over the abdomen and anastomosing with the internal mammary veins and other veins in the axilla.

Case XXX (No. 45899). The second case was that of a young man of 22, of good family and personal history, who had a severe attack of typhoid fever at the age of 17. On the 18th or 19th day of the disease, just before the temperature reached the normal point, there was severe pain in the left groin. A few days later, the temperature being normal, the patient got out of bed and walked about, committing also various indiscretions in diet. This was followed by a relapse, immediately after the onset of which there was swelling, redness and pain in the left groin. About a week after this there was pain and marked swelling of his right leg. There was almost complete loss of power, and it was not until four months later that he was able to go back to work. He had to use crutches for a month. In the erect posture, the legs were considerably swollen; great varicosities developed, and three ulcers appeared on the left leg. Varicosities in the abdomen appeared with convalescence from his relapse. The patient entered the hospital on the eighth of March, 1904, complaining of swelling and ulceration of the legs and abdominal varicosities.

Case XXX (No. 45899) of double iliac thrombosis with extensive abdominal varices.

There was great distension and tortuosity of the veins of both legs and thighs, extending up over the abdomen and anastomosing with branches of the internal mammary veins. Large tortuous veins also extended up into the axillæ. The left leg was everywhere a little larger than the right. The accompanying photograph illustrates well the remarkable character of the varices and anastomoses.

After history of two cases of popliteal thrombosis.—Two cases of popliteal thrombosis were seen several years after the onset of the complication. One of these patients had suffered no inconvenience whatever. In the other, the legs are somewhat weak and toward the end of the day, they are,

at times œdematous. In both instances the affected leg is somewhat larger than the other, but in neither are there marked varicosities.

After history of two cases of thrombosis of the veins of the calf.—In one case, after leaving the hospital, there was considerable œdema with the development of several ulcers. At the present time there is a well marked varicosity of the veins of the inner side of the leg and in the popliteal space. The other patient was communicated with by letter. He states that the "leg from the knee down is the source of a little continuous annoyance; it itches and is frequently inflamed during the warm months. It often goes to sleep." The calf measures an inch and a half more than the other.

(7) Subsequent History of a Case of Typhoid Arteritis with Possible Parietal Thrombosis.

J. H. T. (*Case XVII*, page —), whose attack of arteritis of the left femoral with possible parietal thrombosis, has been previously described, returned on the 27th of March, 1904, in answer to a letter. He considered himself in good health and looked extremely well. He had recently entered the U S. Marine Corps. On inquiry, however, a history of typical intermittent claudication was obtained. "I am," he said, "standing or walking and all of a sudden, it" (the left leg) "gives way on me and I kind of fall." Several times when he has walked too much, his left leg has suddenly given way so that he has actually fallen down. At times, when, he tries to use it, there are sudden pains which shoot upward through his leg. Once when, as a printer, he was lifting a heavy form, the leg suddenly doubled up under him and the form fell upon his toe.

On examination the left thigh and leg were everywhere slightly larger than the right. The femoral pulsation appeared to be equal on both sides. In the popliteal, the pulsation was difficult to feel on either side. The pulsations of the dorsalis pedis were essentially equal. The posterior tibial pulsation, however, was very feeble upon the left as compared with that upon the right.

It is unnecessary here to refer again to the results of cerebral thrombosis.

V. Conclusions.

In considering this series of observations what information have we gained and what conclusions are we justified in drawing?

(1) Typhoid fever is a disease which, from a clinical standpoint, is often associated with symptoms suggestive of a grave weakening of the heart muscle. These changes, whether due primarily to direct action of the typhoid poison on the heart or to impaired nourishment from vaso-motor paralysis, result, in a considerable proportion of cases, in a temporary insufficiency of the mitral valve as indicated by the appearance of apical systolic murmurs which are, not infrequently, transmitted to the axilla. These murmurs develop especially at the height of the disease, during the latter part of the first and in the second, third and fourth weeks, and disappear usually with convalescence. Sometimes, however, they may persist.

12 out of 188 cases of typhoid fever who were followed from three months to fourteen years after convalescence, showed conditions suggestive of organic cardiac lesion. In the majority of these cases a systolic apical murmur had been detected during their illness.

Over one-fifth of our old typhoids in whom, during their illness, a systolic apical murmur was heard, showed on subsequent examination, evidence of organic disease.

The average systolic blood pressure (Riva Rocci) was higher in every decade among our old typhoids than in healthy individuals who had never had the disease.

The radial arteries were palpable with strikingly greater frequency in our old typhoids than in healthy individuals of the same age who had never had typhoid fever.

(2) Endocarditis, while not a common complication of typhoid fever, is probably more frequent than generally supposed. It was present without being suspected, in 3 out of 95 cases coming to necropsy at the Johns Hopkins Hospital, while in 3 further cases out of the remaining 1363, the clinical symptoms suggested its presence.

In the 3 cases discovered at necropsy, the lesion was due to a secondary mixed infection; in 2 instances, staphylococcus aureus; in one staphylococcus albus. It is not impossible that endocarditis is more frequent than indicated by these figures. Out of 188 old typhoids, 8 showed mitral insufficiency; one aortic insufficiency; one, possible mitral stenosis.

(3) Pericarditis is an unusual and unimportant complication of typhoid fever. Three instances only, were noted in our 1458 cases.

(4) Phlebitis and venous thrombosis is a frequent complication of typhoid fever, occurring in over 2.6 per cent of our cases.

The onset occurs usually in the third week or later, and is in most cases, associated with fever, leucocytosis and local pain. Not infrequently the fever and leucocytosis may precede the localizing symptoms.

The complication was ushered in by or associated with chills in 28.9 per cent of our cases.

Chills otherwise unexplained, occurring during the latter part of typhoid fever or in the early stages of convalescence, especially if associated with a leucocytosis, should always suggest the possibility of a developing phlebitis.

The phlebitis is, in the great majority of cases, localized in vessels of the lower extremity, especially on the left side, and is particularly frequent in the femoral veins—about one-half of our cases.

Thrombosis of the iliac and femoral veins is always a serious complication. Although the immediate dangers—gangrene, extension of the thrombus, pulmonary embolism—are not great, the after results are often grave.

In thrombosis of the femoral or iliac vein the affected extremity is always considerably and permanently enlarged,

and there is usually more or less persisting disability—extensive varicosities, often resulting in ulceration; marked weakness of the limb; frequent cramps in the muscles, especially at night and after over-exertion.

(5) Arteritis and arterial thrombosis is a more frequent complication of typhoid fever than is generally recognized.

This complication appears to be especially common in the cerebral vessels although it may occur in the extremities.

The onset may occur at the height of the disease but is commoner in the third week or later. As in the case of phlebitis, the attack is often ushered in by fever and leucocytosis.

In the extremities arterial thrombosis is commonly followed by gangrene; in the cerebral vessels by hemiplegia.

Arteritis in the extremities may be associated with partial parietal thrombosis from which nearly complete recovery may occur. In one of our instances the patient, a year and a half after his fever, showed characteristic symptoms of intermittent claudication.

(6) A survey of our pathological material would suggest that typhoid fever may be a not infrequent cause of focal arterio-sclerotic changes.

In 21 out of 52 cases in which notes upon the condition of the aorta were made there were evidences of fresh endarteritis.

In 13 out of 62 cases in which the condition of the coronary arteries was recorded like changes were found.

This idea is further supported by the remarkable frequency with which sclerosis of the peripheral vessels was found in our old typhoids.

(7) While the deleterious influence of typhoid fever upon the cardio-vascular system is not as great as that of acute rheumatism, yet through the unfortunate frequency of the disease in this country, it is probable that post-typhoid cardio-vascular defects are not uncommon. It would be wise for the practising physician to bear this in mind and wherever possible, to keep his typhoid patients under observation for several years following the disease.

NOTES ON NEW BOOKS.

The Practical Medicine Series of Year-Books. Vol. VI, May, 1904: General Medicine. Edited by FRANK BILLINGS, M. D., and J. H. SALISBURY, M. D. (*Chicago: The Year-book Publishers, 1904.*) Pp. 330.

This volume presents a well-systematized and judiciously selected condensation of the literature of the twelve months preceding its date, in the field of internal medicine. The new work that appeared during that time is presented without comment, and while few of the original articles abstracted suffer more than is necessary in the process of abbreviation, they all hang but loosely together. That compilations of this or similar sort are becoming yearly more indispensable to the busy practitioner, who would keep abreast of the times, is evidenced by the popularity which such works have already attained, even if it were not sufficiently indicated by the truly formidable size of the monthly parts of the *Index Medicus*. To one who has any "Trieb" towards the careful following of special subjects in the current journals, in addition to the call of his practical duty, these compends are both labor-saving and stimulating, enabling him to maintain an intelligent sympathy with workers whose opportunities are different from, or less limited than, his own. As a supplement to the *Index Medicus*—as an amplification of its bare and sometimes misleading titles—the Year-book and others of its class may even be of assistance to the more careful student in his search of the literature for special points of information.

But we should prefer a more synthetic method than has been employed. Unlike some similar publications, the editing of the Year-book has included no evaluation of the material presented; abstract follows abstract without any other relation than that of a common subject. More information of value could be given in a well-conceived critical review of one-half the length. However, the articles are in the main well chosen, and there is little lumber. The references should be more complete, and should follow the system of the Index Catalogue; but so far as we have had opportunity to verify them, they are correct. There is a good index. The paper and presswork are sufficient, considering the necessarily short life of usefulness of works of this class.

The Clinical Study of Blood-Pressure: a Guide to the Use of the Sphygmomanometer in Medical, Surgical, and Obstetrical Practice, with a Summary of the Experimental and Clinical Facts Relating to the Blood-Pressure in Health and Disease. By THEODORE C. JANEWAY, M. D. Pp. XIII; 300. (*New York: D. Appleton & Co., 1904.*)

Dr. Janeway has accomplished a great work, uniting, digesting and grouping the scattered and ill-correlated observations of separate workers in a new field, and bringing their labors before us in an ideal fashion. Neglecting in no detail the ascription of each fact in its order to its proper author, he has recorded everything in the literature of clinical sphygmomanometry that deserves notice, and has drawn amply on the physiologists for criticism and control, making his book complete as a history of our knowledge of the pressure phenomena of the pulse. But far more than this, the author brings a wide personal experience of blood-pressure work to his aid in building up a clear, connected, and reasonable account of the value of the sphygmomanometer in practice, and points out in a way that cannot fail to excite much new and important research, the possibilities as yet undetermined of this, the latest of our instruments of clinical precision. What Wunderlich's great treatise was to clinical thermometry, Janeway's book will prove for clinical sphygmomanometry.

It is only three years since Cushing first brought to this country a Riva-Rocci instrument, and began its systematic use in the surgical wards and operating rooms of this hospital. Dealing with a group of cases that perhaps more than any others are liable to swift and dramatic changes in blood-pressure, Cushing was able to demonstrate the wide utility of the instrument. Since that time Crile, Cabot, and a number of others, have made use of blood-pressure determinations in the clinic, and the new points of attack thus obtained in questions of diagnosis and treatment have led to more detailed studies of the subject than had hitherto been attempted in this country. All this work, as well as that of the earlier and more recent European authors, is fully brought out in the book before us.

Giving up one-third of his space to consideration of the physiological and technical principles involved in the measurement of the blood-pressure in man, Janeway has succeeded in a clear presentation of the most difficult part of his subject. Without going too deeply into the laboratory side of the study of the action of the cardio-vascular mechanism, he has brought out in emphatic relief the physiological fundamentals of knowledge, without which the sphygmomanometer is but a broken reed to lean on in the clinic, and its use unjustified. If any one part of the present work is essential, it is the first section, for the failures

and disappointments that we have seen in the study of pulse phenomena by this instrument have in nearly all instances been due to ignorance on the part of the observer of the usual sources of error, whether in the instrument itself or in the interpretation of its figures.

The various sphygmomanometers in common use are described in detail, and Janeway illustrates a new one of his own design, which, except in one regard, is decidedly the best yet devised for bedside use. Its one drawback is that it is not conveniently portable, and for general use outside of hospitals, we still prefer Cook's modification of the original Riva-Rocci type. True, the new instrument is adequate to the determination of minimum, and hence of mean, arterial pressures, as well as to systolic or maximum measurements; but as in the vast majority of instances all the important clinical information can be drawn from a knowledge of the state of, and the changes in, systolic blood-pressure, the Janeway sphygmomanometer is really less useful than a compact one like Cook's. It has been known for some time that the width of the pressure armlet has, within limits, an influence on the height of the readings obtained with this type of instrument; and if conviction were needed in the matter, Janeway has certainly produced it, and shown that no instrument should be used for other than relative readings that does not employ an armlet at least 10 cm. wide.

The clinical portion of the book is fascinating. A mass of material is presented, much of it original, that cannot fail of itself to excite a great interest among internists and general surgeons alike. But it is in the manner of presentation and correlation of this material that the author has excelled, and may be fairly said to have attained a classic level. The system of his work lends clearness and intelligibility to intricate problems; the emphasis of detail is never tiresome, but always just and illuminating; the charts are well chosen and well drawn. The summary at the head of each chapter makes the book easy to read, and gives consistency to one's general view of the subject treated. The index is ample, the literature tables are exhaustive and the references conveniently placed.

The author's views are so correct, his opinions of the disputed and undecided questions are so just, his enthusiasm is so well controlled, that he leaves one no room or opportunity for criticism. It is a good book, well written, on a vitally interesting subject.

Clinical Treatises on the Pathology and Therapy of Disorders of Metabolism and Nutrition. Part III. Membranous Catarrh of the Intestines. By C. VON NOORDEN, with the collaboration of DR. CARL DAPPER. Pp. 64. (*New York: E. B. Treat and Co., 1903.*)

Muco-membranous Entero-colitis: Symptoms, Complications, Etiology, and Treatment. By M. DE LANGENHAGEN, M. D. Pp. VI; 115. (*London: J. and A. Churchill, 1903.*)

A comparative reading of these two little books offers a strong lesson in method and in the value of the open mind. De Langenhagen, who is a resident at Plombières, in reviewing a personal material of over 1200 cases of mucous colic, is led by the ignis fatuus of arthritism to mistaken judgments concerning etiology and treatment, to imperfect analysis of his large series of cases, and to conclusions that lack correlation and the quality of bringing conviction to the reader. The work of v. Noorden and Dapper, on the other hand, based on a group of less than eighty cases, is a model of clinical style and a source of profit and satisfaction in the reading.

Taking a broader view perhaps, but certainly a less accurate one, than that of the two German clinicians, de Langenhagen attributes all but the most exceptional instances of "muco-membranous entero-colitis" to the malign influence of that diathetic abstraction, "neuro-arthritisme," which has so much power over the minds of many French internists. His picture of the disease, though rather overburdened with accessory detail, is accurate and generally clear, but we cannot agree with the author as to the constant concurrence of general enteroptosis with the affection under consideration. It is with regard to the views of causation and treatment expressed that the French brochure is most unsatisfactory. Local treatment of the bowel itself is well discussed; but we do not think that the recommendation of an unirritating diet poor in residue, and of drug control of the attendant constipation, would meet with general favor. The neurotic element in the disease is recognized, and treatment away from home is advised, perhaps not without regard to the author's position at a prominent watering-place, where the various hydrotherapeutic measures recommended can be best carried out.

The translation is atrociously bad, the reader's attention being constantly diverted from the subject-matter by the grotesque picture of Gallic idiom faithfully preserved in the unsympathetic menstruum of the English vocabulary.

The English version of Part III of v. Noorden's series of small handbooks suffers less in translation than does its French companion. The paper and presswork are hardly worthy of the matter which they present, but the form of the book is convenient. About half of its pages are taken up with a consideration of the pathology, and a condensed but ample discussion of the older and the more recent literature. The term "mucous colic," which is used, seems more acceptable than that of mucous colitis as a name for a condition whose actual inflammatory nature is, to say the least, not proven, and it is the one name that most exactly pictures the cardinal symptoms of the disease.

v. Noorden finds that in all cases there has been precedent constipation in persons of a neurotic disposition. But only a few constipated neurasthenics have mucous colic; and the authors are far from recognizing with Boas this highly characteristic symptom complex as simply dependent upon the presence of scybala in the bowel. The link connecting the sluggish action of the bowels with the abnormal general nervous state in the production of the symptom picture, they find in an involvement of the nerve mechanism governing the secretion of mucus in the large intestine, so adopting Nothnagel's postulate of a secretory neurosis. That a sensory intestinal neurosis co-exists, or at least that the mere presence of mucus adhering to the bowel wall causes sensory symptoms of a severity greater than would be the case in persons of a stable nervous constitution, seems admissible.

The therapy suggested, one which has probably been as widely accepted as any, can be summed up in very few words. It consists in coarse diet, and the prohibition of cathartic drugs, with, of course, appropriate measures directed to the general nervous system. The essential feature in treatment should be the relief of the constipation by means of food rich in cellulose—a re-education, as it were, of the bowel wall. High oil enemata are favorably considered in the early stages of the "cure" but the use by rectum of solutions having any irritant properties at all is not encouraged. When on the diet recommended, which is well summarized without iron-bound prescription of detail, daily soft evacuations are obtained, the patient's general condition improves, and as his state of nutrition is bettered the whole symptom-complex disappears. By these methods the authors were able to obtain complete recovery in 79 per cent of their cases—complete and permanent recovery in just one-half of the whole number treated.

Only casual attention is given in these works, written as they are from a purely medical standpoint, to the forms of mucous colic associated with ovarian tumors or disease of the uterine adnexa.

Both of these booklets would be better for even brief and general indexes; the tables of contents do not cover the deficiency.

The Skin and Venereal, Nervous and Mental Diseases. Edited by W. L. BAUM, M. D., and HUGH T. PATRICK, M. D. The Practical Medicine Series of Year-books. Edited by GUSTAVUS P. HEAD, M. D. (*Chicago: The Yearbook Publishers, 1903.*)

The value of this little book lies, of course, in the opportunity it affords of ascertaining at a glance the outlines of the most recently acquired knowledge upon the subjects which it considers, as well as of obtaining correct references as to where such knowledge can be obtained in full without further trouble. Its purpose and scope are so general that it would not be suitable or profitable to review it in detail. All that can be said here is that the items selected for comment are chosen with judgment, and they are discussed in a very satisfactory manner. The compilation is most useful to the busy practitioner.

Epilepsy and its Treatment. By WILLIAM P. SPRATLING, M. D., Superintendent of the Craig Colony for Epileptics at Sonyea, N. Y. Handsome octavo volume of 522 pages, illustrated. (*Philadelphia, New York, London: W. B. Saunders & Company. Baltimore: Medical & Standard Book Co., 1904.*)

This is the first complete treatise on Epilepsy since the appearance of Echeverria's work, published over 33 years ago, and represents the practical experience of Dr. Spratling as Superintendent of the Craig Colony for Epileptics at Sonyea, N. Y., during a period of ten years.

This book is well-printed and well-illustrated and presents an attractive appearance. It contains a wealth of clinical experience presented in such a manner as to command the attention of the general practitioner. It is in fact more a book for the practical man than for the student. It is written in a popular style and is very readable.

In the line of treatment little that is new is presented. The chapter on the results of surgical interference is substantially the same as the one which was published in the *American Journal of Insanity* last year. The ultimate results of all sorts of surgical interference do not seemingly justify the surgeon in promising much benefit. The chapter on the medical treatment presents a similarly discouraging prospect. From a careful reading of the book one is impressed with the conviction that more is to be expected from open-air life, good hygienic conditions, freedom from worry and adjusting the patient to the life of an invalid requiring custodial care and parental guidance rather than medicine or surgical interference.

The descriptions of disease conditions are excellent and the whole book indicates a careful systematic study of the subject. The State of New York has reason to be proud of the character of the work done at Craig Colony. It is evident that here the foundations are being laid for a fuller knowledge of the disease and a scientific mode of treatment.

Burdett's Hospitals and Charities, 1904. Being the Year Book of Philanthropy and the Hospital Annual, containing a review of the position and requirements and chapters on the management, revenue and cost of charities. An exhaustive record of Hospital Work for the Year. By SIR HENRY BURDETT, K. C. B. (*London: The Scientific Press (Limited), 28 & 29 Southampton St., Strand W. C.*)

The most interesting feature of the present Annual is an elaborate discussion of the space requirements of the modern hospital. From this it appears that St. George's Hospital has a site area of 191 superficial feet per patient; Westminster Hospital has 134 feet; The Royal Free Hospital has 253 feet; Middlesex Hospital has 267 feet; St. Mary's Hospital has 136 feet; Charing Cross has 86 feet; University College Hospital has 160 feet; Guy's Hospital has 364 feet; St. Thomas's Hospital has 652 feet, and St. Bartholomew's Hospital, when the recent additions made to the site are utilized, will have 420 feet per bed. The contention of the Annual is that no hospital ought to have less than 450 superficial feet per bed and that consequently St. Thomas's is the only London hospital with a medical school attached which possesses a site which at present fulfils this essential requirement of modern science. The writer adds that "formerly 300 superficial feet per bed were regarded as enough for a town hospital; but the continuous increase in the demands for special departments, and above all for the complete aëration of every building occupied by patients, has produced results which raise the minimum of efficiency to 450 superficial feet per bed. In the case of a virgin site upon which it is possible to plan a hospital and medical school in its entirety, it is possible to so arrange the accommodation in the various buildings that every available foot of the site shall be utilized to the best advantage. In London, owing to the circumstances we have already referred to, with the exception of St. Thomas's Hospital, the medical school buildings have been added from time to time and were probably not contemplated when the hospital buildings were first planned and erected. This accounts, too, for the position in which two of the larger hospitals stand with regard to this question. We have taken out the figures and we find that upwards of 56 per cent of one site is occupied by buildings, although the ward blocks, theatres, etc., occupy less than 16 per cent of the whole available area, against about 20 per cent, or one-fifth of the site which is covered by medical school and kindred buildings. We mention this to show that whereas for a town hospital the unit has been roughly, 100 beds per acre of site, if such a hospital has a medical school attached to it the unit must be increased to something like 50 or, at the outside, 60 beds per acre."

The remedy proposed by the writer is to consolidate the teaching in fundamental branches during the first two years of the medical curriculum and to use hospitals wholly for medical teaching. This will do away with the duplication of buildings and leave the grounds of the hospitals less cumbered with buildings which have no connection with the clinical study of medicine.

The Annual for 1904 is filled with interesting and valuable statistics. It should be in the hands of every hospital officer.

University of Pennsylvania: Contributions from the William Pepper Laboratory of Clinical Medicine. No. 4. (*Philadelphia, 1903.*)

This is a formidable collection of reprints of articles written by the various workers in the Pepper Laboratory. At least one-half is devoted to a series of interesting neurological cases reported by W. G. Spiller. One of the more important is a "Report of Two Cases of Multiple Sclerosis with Necropsy." In America there have only been three such cases reported. The author's first case was clinically and pathologically typical. The second had several unusual features, viz., pronounced muscular atrophy, secondary degeneration of the crossed pyramidal tract, and almost complete internal ophthalmoplegia resulting from sclerosis in the nuclei of the 3d, 4th and 6th nerves.

Another paper by Spiller and Hendrickson on "Multiple Sarcomatoses of the Central Nervous System" is worthy of attention. Two cases are reported at great length and their histological characters clearly depicted. McCarthy, in a paper on the "Formation of Hæmolymph Glands from Adipose Tissue in Men," confirms clinically Warthin's recent experimental work in sheep and goats. Both were cases of Adiposis dolorosa, in the fat of which newly-formed hæmolymph tissue was found. Evans and Sailer report a "Case of Colon Infection Simulating Typhoid Fever." The patient had fever for four weeks, enlarged spleen, rose spots, and a mild relapse. The serum agglutination test was negative to *B. Typhosus* and four strains of *B. Paratyphosus*, but positive to *B. Coli communis* in high dilutions (1:150) in one hour. A

control with four typhoid sera was negative. The more absolute proof—the isolation of *B. Coli* from the blood—was wanting.

Stengel and White report a remarkable case of "Chronic Acetanilid Poisoning with marked alterations in the Blood." The patient was a girl of 25 years who for four years had been suffering with infradental neuralgia, for which she had taken chloral, hyoscyamine, as well as acetanilid in large doses over a period of three years. She had marked cyanosis, cardiac, hepatic, and splenic enlargement, but no dyspnœa or œdema. The blood showed a marked secondary anæmia with great numbers of nucleated red cells (25,000 to 75,000 per cmm.), of which 91.4 per cent were normaloblasts, and 3.5 per cent megaloblasts. With the withdrawal of the drug the patient made a complete recovery.

Medical and Surgical Report of the Presbyterian Hospital in the City of New York. Vol. VI. January, 1904. Edited by ANDREW J. MCCOSH and W. GILMAN THOMPSON. (*New York: Trow Directory Printing and Bookbinding Company.*)

This compact volume contains many useful contributions, and is a credit to the institution which gave it birth. The first article is a statistical study, by Tuttle and Carter, entitled "Data regarding Acute Lobar Pneumonia" at the Presbyterian. In a period of about six years 600 cases of acute lobar pneumonia were treated (as compared with 8470 admissions), with a mortality of 34.8 per cent. Two-thirds of the cases occurred from December to May inclusive. It is interesting to note that 18 cases were complicated by empyema, with one death. More than 12 per cent received *no* medical treatment whatever, while of the remainder the great majority received only symptomatic treatment. Ellsworth Eliot, in an article entitled "The behavior of the Costal Arch in Diseases of the Abdominal Organ," describes a new method of examination—the determination of the resistance of the costal arch by means of palpation, with one hand on either half of the arch. He finds that the resistance can always be elicited in acute and subacute processes of the contiguous organs in the upper half of the abdomen, and that the degree is in direct ratio to the intensity of the process.

John Howland gives a résumé of "The Pathological Anatomy of the Shiga Bacillus Infection of the Intestines in Infants." Among 32 autopsies from three of the New York hospitals, the organism was found (either clinically or post-mortem) in the great majority, and proved to belong to the "Harris" or "Flexner-Manila" type, i. e., it fermented mannite with acid formation. "A contribution to Cyto-Diagnosis in Pleural Effusion with especial reference to the Tubercular Form," by H. S. Carter, is, in our opinion, the most important article in the book. After a study of 36 cases (10 of effusion of undoubted tubercular origin, 19 of probable tubercular origin, 3 of serous effusion after pneumonia, and 4 of transudation in cardiac or renal disease), he concludes (1) that a true pleural effusion has a specific gravity over 1010, a high fibrin and albumin content in contrast with a pleural transudate (2) that an absolute diagnosis of tubercular pleurisy can be made by a morphological examination of the fluid, and rests upon a high specific gravity (1012-1024), a high amount of fibrin and albumin with an accompanying lymphocytosis.

Ingen and Frissell report "A House Epidemic of Paratyphoid Fever," in one of which the organism was isolated post-mortem. The details of the autopsy are interesting as the case showed no apparent change in Peyer's patches, but several punched-out ulcers near the iliocæcal valve.

The last article is a "Report on Malignant Endocarditis, with a collection of Thirty-two Cases," by M. H. Sicard. There were six cases due to the staphylococcus, three to the pneumococcus, and two to the streptococcus, in all of which the organism was isolated either during life from the blood, or at autopsy. The remaining twenty-one cases did not show an organism, but the diagnosis was based upon other data. Of the 32 cases, 8 occurred coincidently with rheumatism and 7 gave a previous history of such, so that endocarditis was a pre-existing factor in the majority. Among 19 autopsies there were 16 with emboli. Hæmorrhage occurred in 16 cases; and a leucocytosis of at least 15,000 per cmm. in all but one case.

International Clinics: A Quarterly of Illustrated Clinical Lectures and Especially Prepared Original Articles on Treatment, Medicine, Surgery, etc., by leading members of the Medical Profession. Edited by A. O. J. KELLEY, Philadelphia. Vol. I. Fourteenth series, 1904. (*Philadelphia: J. B. Lippincott Company.*)

Space does not permit even a cursory review of each interesting and instructive paper that is included in this volume. Only the most important will receive attention.

In the section on Treatment, by far the most important paper is that by Widal and Javal of Paris, "The Chlorid Reduction Treatment of Parenchymatous Nephritis." The authors show that the degree of albuminuria follows the oscillations of the chloride retention curve, and that the tissue hydration and dehydration follow the giving or withdrawing of chlorides. A chloride reduction must be insisted upon at certain times in the diet of cases with parenchymatous nephritis. This can be arrived at by means of a pure milk or even a meat diet *lacking in chlorides*. Further, they emphasize that the determination of the amount of chlorides excreted is of no value, except in relation to the chlorides absorbed. In addition, they show that albuminoids of animal origin (as of milk) are less harmful than those of meat, because of the salt that is added to the latter.

In a "Clinical Study of Adonidin," R. W. Wilcox shows that it is indicated in cardiac disease, in which a rapid pulse of low tension is a notable feature, and that the drug possesses certain advantages over digitalis,—its action being more permanent and less vaso-constricting than the latter.

The article entitled "The Therapeutic Application of Colloid Silver," is interesting but not convincing. More experimental work has yet to be done and some rational explantion for its action offered before the exhibition of the drug will receive the support of the profession.

Two papers on the treatment of neurasthenia in general, and of gastric neurasthenia in particular, bring forward nothing new. The author of the latter seems to overvalue electricity as a therapeutic agent.

Davis, of Chicago, in the section on medicine points out that "the increased prevalence and mortality of pneumonia during the last sixty years," is due not to any climatic or dietetic change, but to the lowered resistance of the community from a more general use of alcohol and migration of youth of both sexes from country to town. In the outline of the various methods of treatment, he deplores the use of "alcoholic and other anæsthetic drugs" in the course of the disease.

Walsh, of New York, in "The Early Diagnosis of Pulmonary Tuberculosis," emphasizes the importance of inspection and auscultation of the chest, the rapidity of the pulse, and the persistent slight loss in weight—data which enable one to make a diagnosis even in the absence of bacilli in the sputum.

The section on Surgery contains a paper by Beck, of Chicago, on "Angioma and its treatment," and also discusses the pathology and etiology of these tumors; he makes a plea for their radical treatment by extirpation. Of more general interest are the papers on "Intestinal Anastomosis" and "The Complications met in the Surgical treatment of Diseases of the Testicle."

The section on Gynecology is devoted, strangely enough, to two papers on "the Non-operative treatment of Inflammations of the Genital Tract" and "of Chronic Ovarian Lesions."

In the section on the "Progress of Medicine," the literature

of the year is reviewed, and the more important contributions are detailed. Thus, the importance of the house fly in dissemination of typhoid fever was emphasized by Hamilton and Turner; the bacterial origin of acute rheumatism is supported by Ainley Walker and F. Meyer, but contradicted by C. Philipp, who believes that rheumatism is a "morbus sui generis." R. L. Jones reports a series of cases in which Graves' disease and arthritis deformans occurred together, and believes that a definite relation exists between the two.

The section on the "Progress of Surgery" by Bloodgood is very exhaustive and merits a careful perusal, especially the article on the Surgery of the Stomach. Surgery has been making tremendous strides in the treatment of simple ulcer and cancer of the stomach, thanks to such men as Mikulicz of Germany, Moynihan and Robson of England, and the Mayos of America. Operative interference for gastrorrhagia is considered more justifiable than formerly, and the operation of choice is a gastro-enterostomy, thereby procuring physiological rest and so affording an opportunity for the healing of the ulcer. A résumé of Brunner's communication on "Gastric Perforation" receives especial attention. The plates illustrating the article on Cancer of the Stomach are very beautiful and instructive.

The section on "Treatment," by Stevens, includes an epitome of the various drugs and antitoxins in vogue, and the special indications for their use.

C. P. Howard.

BOOKS RECEIVED.

Scientific Memoirs. By the Officers of the Medical and Sanitary Departments of the Government of India. (New Series.) No. 7. *Some Observations on the Poison of the Banded Krait (Bungarus Fasciatus).* By Captain George Lamb, M. D. (Glag.), I. M. S. Issued under the Authority of the Government of India, by the Sanitary Commissioner with the Government of India, Simla. 1904. 4to. 32 pages. Office of the Superintendent of Government Printing, Calcutta.

A System of Physiologic Therapeutics. Edited by Solomon Solis Cohen, A. M., M. D. Volume VIII, *Rest, Mental Therapeutics, Suggestion.* By Francis X. Dercum, M. D., Ph. D. 1904. 8vo. 332 pages. P. Blackiston's Son & Co., Philadelphia.

The Therapeutics of Mineral Springs and Climates. By I. Burney Yeo, M. D., F. R. C. P. 1904. 12mo. 760 pages. W. T. Keener & Co., Chicago.

Saint Thomas's Hospital Reports. New Series. Edited by Dr. H. P. Hawkins and Mr. W. H. Battle. Volume XXXI, 1902. 8vo. 136 + 405 pages. 1904. J. & A. Churchill, London.

Progressive Medicine. A Quarterly Digest of Advances, Discoveries, and Improvements in the Medical and Surgical Sciences. Edited by Hobart Amory Hare, M. D., assisted by H. R. M. Landis, M. D. March 1, 1904. 8vo. 337 pages. Lea Brothers & Co., Philadelphia and New York.

Manual of Materia Medica and Pharmacy. Specially Designed for the Use of Practitioners and Medical, Pharmaceutical, Dental, and Veterinary Students. By E. Stanton Muir, Ph. G., V. M. D. Third edition, revised and enlarged. 1904. 8vo. 192 pages. F. A. Davis Company, Philadelphia.

The Infectivity of Enteric Fever. With Observations on its Origin and Incidence at Caius College, Cambridge, Festiniog and Wicken-Bonant. By Alexander Collie, M. D. (Aberd.). 1904. 8vo. 47 pages. John Wright & Co., Bristol; Simpkin, Marshall, Hamilton, Kent & Co., Ltd., London.

Orthman's Handbook of Gynæcological Pathology. For Practitioners and Students. Translated by C. Hubert Roberts, M. D. Lond., F. R. C. S. Eng., M. R. C. P., assisted by Max L. Trechmann, F. R. C. S. Eng., M. B., C. M. Edin. 1904. 8 vo. 127 pages. John Bale, Sons & Danielsson, Ltd., London.

A Practical Treatise on Medical Diagnosis. For Students and Physicians. By John H. Musser, M. D. Fifth edition, revised and enlarged. Illustrated with 395 wood-cuts and 63 colored plates. 1904. 8vo. 1213 pages. Lea Brothers & Co., Philadelphia and New York.

Scientific Memoirs. By the Officers of the Medical and Sanitary Departments of the Government of India. (New Series.) No. 8. *A Preliminary Report on a Parasite Found in Persons Suffering from Enlargement of the Spleen in India.* By Lieut. S. R. Christophers, M. B., I. M. S. Issued under the Authority of the Government of India by the Sanitary Commissioner with the Government of India, Simla. 1904. 4to. 17 pages. Office of the Superintendent of Government Printing, Calcutta.

THE JOHNS HOPKINS HOSPITAL BULLETIN.

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